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EDITORIAL

NON-OPERABLE GROUP OF CRANIOCEREBRAL INJURIES

WE are inclined, in present day methods of teaching surgery, to resort considerably to groupings and classifications of disease entities and pathologic pictures, and occasionally merely allude to the fact that in some conditions, one clinical picture "overlaps the other." A clearer understanding of the pathogenesis will undoubtedly be followed by a more accurate knowledge of the pathologic findings, and this in turn will make more easily understandable the symptomatology. With meticulous pseudoprecision, for example, we teach and learn the classifications of brain injuries, and ascribe to each entity a group of pathologic findings and the resultant symptomatology, as though each had its own classical story. In approximate order of increasing severity, we group the non-operable craniocerebral injuries as concussion, edema and congestion, and contusion and laceration.

The pathology of concussion is not definitely known. Perhaps it is a reflex of some type resulting in anoxemia. The chief symptom, however, is unconsciousness for a short period of time, followed by complete recovery.

The pathology of edema and congestion in themselves is rather difficult to evaluate, as edema and congestion are rarely present without the accompaniment of more serious brain damage. It might perhaps be briefly stated that there is here an increase in brain volume, which if sufficiently sustained, eventuates in intracortical hemorrhage. The signs and symptoms, as is well known, are unconsciousness followed by a return of consciousness, after which there is headache and dizziness, nausea, and some loss of memory. There is often increased intracranial pressure, but the cerebrospinal fluid is normal.

In contusion, there is some injury to the brain surface with torn cortical vessels. In laceration, the cortical surface is actually torn. Here the symptoms are more profound. There is a definite and prolonged period of unconsciousness, which may last for days. There is nausea and vomiting, and headache. In the more severe cases, there may be dilatation of the pupils, irregularity of respiration, and increase in pulse and temperature. Surgical shock is frequently present. The intracranial pressure is high, and the cerebrospinal fluid contains blood.

It is thus quite obvious that the consideration of the pathology of contusion and laceration includes that also occurring in edema and congestion, as well as that of concussion. The same can be said as regards symptomatology. Both contusion and laceration are associated with edema and congestion, and these in turn incor-

porate concussion. Stating it in another way, following craniocerebral injuries, edema and congestion include concussion. For example, the unconsciousness in laceration is not necessarily due to the tear in the brain cortex, as the concussion present was sufficient to cause it; the more severe pathologic findings and symptoms · are due to the added damage, i.e., from the arbitrary division beyond concussion.

It is thus seen that the present classification is more of a didactic rather than either a true clinical or pathologic one. If the foregoing is kept in mind, there will be a better understanding and appreciation of some of the non-operable craniocerebral injuries, and more important, a better rationalization when thinking in terms of therapeutic measures.

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ORIGINAL ARTICLES

POSTOPERATIVE WOUND DISRUPTION AND EVISCERATION

AN ANALYSIS OF THIRTY-FOUR CASES WITH A REVIEW OF THE LITERATURE

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THIS study includes thirty-four cases of postoperative eviscerations that have occurred in the Cedars of Lebanon Hospital, Los Angeles, California, together with remarks based on a review of the recent literature with the object of analyzing the mechanism of production and suggestions in the prevention and treatment.

A review of the literature is a comedy of contradictions. It is instructive because various authors have arrived at different conclusions based on their individual series of statistics. It teaches us to be cautious in drawing conclusions from any one single series.

In this study we shall discuss (1) the factors which are inherent in the patient, such as age, sex, primary disease, previous operations, preoperative condition, postoperative course; and (2) the factors in the operation and postoperative course for which the surgeon is responsible, such as anesthesia, type of incision, closure, operative procedure, infection, and drainage. Certain inherent factors ascribed to the patient which have a bearing on the incidence of evisceration may be partially diminished by the surgeon, e.g., (1) preoperative—emaciation, dehydration, anemia and shock; (2) postoperative—distention, cough, vomiting, hiccough and restlessness.

Then we shall acquaint ourselves with the materials with which we are working, viz., tissues, wound healing and catgut, later discussing the mechanism of eviscera-tion, reviewing preventive steps in the treatment adopted by various operators and summarize our conclusions based on a review of the literature.

Incidence. An idea of the incidence will be gleaned by listing the sources from which analyses and comparisons have been made in this study. Sokolov[60] analyzed the

TABLE I

INCIDENCE AND MORTALITY OF DISRUPTIONS AND EVISCERATIONS

Author	Laparotomies	Disruptions and Eviscerations	Per Cent	Deaths	Per Cent Mortality
Bettman and Lichtenstein		32	0 43	12	38
Bowen		34		17	50
Colp	2,750	26	0 9	8	28
Eggers		20		6	30
Eliason and McLoughlin	9,155	25	0 27	8	32
Fallis	7,903	50	0 64	17	34
Grace		46		15	39
Glenn and Moore	2,927	22	0 75	10	45 5
Glasser	3,234	8	0 24	6	75
Heyd	3,145	8	0 25	2	25
Hinton	621	19	3 05	3	16
Horner	9,155	3	0 29	0	0
Jenkins		36		13	36
Koster and Kasman		17	0 22	3	18
Maes, Boyce and Mc-Fetridge	.	44		13	30
Meleney and Howes		55	1	22	44
Milbert	1,560	20	1 3	11	55
Singleton and Blocker	0,000	61	0 67	19	31 15
Starr and Nason	2,455	15	0 61		
Totten	19,473	47	0 24	19	40
White		34		17	50

replies which he received from 1,000 questionnaires sent out to surgeons all over Europe and concluded that the incidence varied from 2 to 3 per cent of all abdominal operations. Carl Eggers[12] (1934) showed

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from the records of Lenox Hill Hospital ten cases in the last five years. (This does not include private cases.) Maes, Boyce and McFetridge[43] (1934) report forty-four cases which occurred in the last ten years (1924–1934) in New Orleans Charity Hospital. Grace[20] (1934) reports thirty-six cases from the records of First Surgical Division of Bellevue Hospital for the past fifteen years. Colp[10] (1934) reports twenty-six cases from a series of 2,750 consecutive laparotomies from Surgical and Gynecological Services of Mount Sinai Hospital. Meleney and Howes[45] report fifty-five cases (incidence of 1 per cent) from Surgical Service of the Presbyterian Hospital in New York in the eight years from 1926 to 1934. White[66] (1934) reports thirty cases from the Roosevelt Hospital, New York. Heyd[25] (1934) reports four cases in 2,125 personal cases, and four in one year in 1,000 consecutive clinic cases. In 7,903 consecutive laparotomies performed at the Henry Ford Hospital, Fallis[15] (1937) reported fifty wound disruptures—an incidence of 0.64 per cent. Glenn[19] (1937), analyzing, 2,927 abdominal operations, found twenty-two cases of evisceration—an incidence of 0.75 per cent. From a total of 19,473 consecutive abdominal operations performed at the Los Angeles County Hospital, Totten[63] (1938) reported forty-seven wound ruptures—an incidence of 0.24 per cent.

Age. No period of life appears to be exempt. The greatest incidence in our series was in the fourth and fifth decades. This is to be expected since it is during this twenty year period that most surgical operations are done and one in which abdominal carcinoma is more frequent and subject to exploration. In our series the youngest was 10 (splenomegaly), the oldest 68 (carcinoma of stomach). Jenkins,[30] reviewing 1,294 reported American and European eviscerations, found the average age to be 44 years, and in his reported series of thirty-six cases, three were infants 2 to 4 months old.

Sex. Sokolov collected and reviewed 730 eviscerations and found that it occurred twice as frequently in men as in women. In the fifty-five cases reported by Meleney and Howes there were thirty-six males and fourteen females. In contrast, Madelung[42] reporting 157 eviscerations from a European clinic, states that wound rupture occurs three times as commonly in women as in men. Colp in his twenty-six cases reports male fourteen, female twelve, a ratio of 7 to 5. Maes, Boyce and McFetridge in their forty-four cases, report twenty-one male, twenty-three female. In Jenkins' summary of 1,294 cases, 649 were in males (57 per cent); 482 (43 per cent) in females. *In our series* fourteen were female, twenty male.

Seasonal Variation. Sokolov was induced to believe that the season of the year exerted a direct influence on the incidence of wound evisceration. His statistics proved that most wound separations occurred during the winter and spring months. Colp in his twenty-nine cases found no seasonal relationship. *In our series* the highest incidence was in summer, the lowest incidence in the fall.

Mortality. In the cases reviewed the mortality varied from 16 to 75 per cent. Colp gives 28 per cent; Meleney and Howes, reporting forty cases, give a mortality of 44 per cent; Grace, reporting thirty-six cases, gives a mortality of 41 per cent, and White, reporting thirty cases, gives a mortality of 53 per cent; Glenn[19] (twenty-two cases) 45 per cent; Fallis (forty-nine cases) 34 per cent; Milbert (twenty cases) 54 per cent. There were thirteen deaths in Jenkin's series of thirty-six cases—a mortality of 36 per cent; and in his analysis of 1,294 cases, the average mortality was 35 per cent.

In our series of thirty-four cases, seventeen recovered, seventeen died, giving a 50 per cent mortality. Of the seventeen who died, nine had carcinoma; of the seventeen who recovered, three had carcinoma. Analysis of these cases makes it quite clear that most of them did not die because their wounds ruptured, although it is correct to assume that many times

the accident precipitated the fatality. Many of the cases would have terminated fatally even if the complication had not occurred.

Primary Diseases and Evisceration. Analysis was made to see if there was any relation between the diseases for which the patient seeks surgery and the incidence of evisceration. While the number of cases is small, the frequency of evisceration in certain conditions was of interest. *In our series* of thirty-four cases, twelve patients had carcinoma, of whom nine died and three recovered. Of the nine that died carcinoma was present as follows: generalized, one; pancreas, one; stomach, four; intestine, three. In the three that recovered carcinoma was present as follows: rectum, one; stomach, one; ovary, one. Seven had ulcers of the stomach or duodenum, four had gall-bladder disease, three had fibroids, three had appendicitis (two acute and one chronic), one had an ovarian cyst, one splenomegaly, one a ventral hernia, and two pregnancies requiring cesarean section.

In Colp's series, malignant tumors were responsible for 28 per cent of eviscerations, inflammatory diseases of bile passages for 23 per cent, gynecological diseases, as fibroids, 19 per cent. Starr and Nason,[61] in 2,245 laparotomies, had fifteen eviscerations or 0.61 per cent, and it is interesting to note that 40 per cent of these occurred following operations for carcinoma; that out of 135 laparotomies for carcinoma the incidence for evisceration was 4.4 per cent, again emphasizing the increased frequency of evisceration in this condition.

In Jenkin's series of thirty-six cases, nineteen had abdominal malignancies; of the nineteen malignancies there were ten carcinomas of the stomach, four of the rectum, three of the pancreas, and two of the colon. In the summary of data on 1,294 eviscerations Jenkins found 296 or 25 per cent followed operation for malignancies, 12 per cent following biliary tract surgery, and 18 per cent following gynecologic procedures. Meleney and Howes, in their series of fifty cases, showed the great-

est incidence following biliary and gastric surgery, sixteen cases followed gastric surgery, fourteen cases followed biliary surgery. Malignant disease was responsible for 27 per cent of Glenn's series of twenty-two cases.

TABLE II

INCIDENCE OF MALIGNANCY IN DISRUPTED AND EVISCERATED CASES

Author	Disruption and Evisceration	Carcinoma	Per Cent
Bettman and Lichtenstein	32	6	19
Colp	29	8	27 6
Eliason and McLoughlin.	25	6	24
Fallis	50	8	16 3
Glasser	8	0	0
Glenn and Moore	22	6	27
Grace	46	10	21 8
Jenkins	36	19	53
Koster and Kasman	17	2	11 8
Maes, Boyce and McFetridge	44	5	11 4
Meleney and Howes	56	19	34
Milbert	20	8	40
Singleton and Blocker	61	4	6 5
Starr and Nason	15	6	40
Totten	47	7	15
White	30	5	16 6
Bowen	34	12	35 2
Total or average	572	131	23 5

TABLE III

INCIDENCE OF DISRUPTIONS AND EVISCERATIONS IN MALIGNANT DISEASES

Author	Laparotomies in Malignancies	Disruptions and Eviscerations	Per Cent Incidence	Laparotomies	Per Cent Incidence
Colp	316	7	1 20	2,750	0 9
Glenn and Moore	582	6	2 2	2,927	0 75
Starr and Nason	135	6	4 4	2,455	0 61

It is apparent that the largest group of cases occurred in patients having carcinoma of an abdominal viscus. This tends to confirm the usual surgical impression that in diseases accompanied by cachexia,

the wound tends to heal more slowly and when these are subjected to increased intra-abdominal pressure, the wound frequently gives way. Certain benign growths such as fibroids are often attended with harmful systemic effects, as for example hemorrhage and cardiac decompensation.

The frequency also of evisceration in patients with peptic ulcers and gall-bladder disease is due to the factors of vomiting, hiccough, distention, infection and associated liver pathology rather than to the patient's state of nutrition. Statistics from the literature agree with our findings.

In our series, evisceration most frequently followed operations on the stomach. Eviscerations followed stomach surgery (ten times); gynecological and obstetrical surgery (seven); cholecystectomy (five); intestinal surgery (four); appendectomy (four); exploratory laparotomies for inoperable carcinoma (three); splenectomy (one).

In contrast to the frequency in our series of eviscerations in carcinoma, gastric ulcer and gall-bladder disease, we have the figures from Maes, Boyce and McFetridge's forty-four cases representing a period of ten years at the New Orleans Charity Hospital in which 50 per cent of their incidence was in cases of appendicitis. Ten of the twenty cases were drained. Still more confusion is added to this group because of the fact that in six of these the McBurney incision was used after which evisceration is seldom reported.

Previous Operations. Operations performed through previous scars have the reputation of being more likely to separate than those of a primary incision. Colp, in his series of twenty-six cases, reports one case of evisceration following secondary operation. He reports a series of sixty-one patients reoperated on through previous incisions without a single case of evisceration.

In our series it occurred in five cases in which previous scars were excised. One of these five patients had had nine previous abdominal operations. Another one eviscerated following two successive operations. On December 19, 1931 he was operated on for perforated duodenal ulcer and eviscerated six days later. One year later on December 6, 1932, he was operated for intestinal obstruction and eviscerated three days postoperatively. Multiple operations prolong the operating time, increase the trauma, necessitate enlarging the incision, vigorous traction—which may be contributing factors in the etiology of disruption. In Fallis' series of forty-nine eviscerations, thirty-six or 75 per cent, were subjected to multiple operations.

Preoperative Condition. In twelve cases the preoperative condition was good; in eighteen cases the preoperative condition was poor; two had active tuberculosis; one had had asthma for twenty-five years; five were obese; two had anemia requiring transfusion; two were emaciated; two dehydrated, and two had myocardial damage. In two cases, the history was incomplete as to preoperative conditions. Therefore, in more than one-half of the patients who eviscerated the preoperative condition was complicated by diseases named above.

Postoperative Course. The postoperative course of the eviscerated cases was analyzed to determine the extent to which postoperative complications were contributing or exciting factors in the production of evisceration. It must be admitted that in some the evisceration was the result of postoperative vomiting, hiccoughing, and distention. In other cases it was the cause of these complications. In only thirteen of the eviscerated cases was the postoperative course smooth.

The serious rôle which pulmonary complications play in abdominal surgery is well known and the part which they might play as the precipitating factors in evisceration is obvious.

Respiratory infections were the exciting agents in nine of the thirty-four eviscerations: asthmatic (two); bronchial pneumonia and later positive sputum for

tuberculosis (one); pleural effusion (three); bronchial pneumonia (one); and cough (two).

Persisting vomiting and distention requiring lavage and Connell suction were noted in thirteen cases. While vomiting usually precedes evisceration, one cannot lose sight of the fact that it may also be the result of partial obstruction of the eviscerated gut. Four patients precipitated their evisceration by undue exertion in the early postoperative days. One fell out of bed eight days postoperatively. One got out of bed three days postoperatively, one five days and a third six days postoperatively.

Persistent hiccoughing was noted in two cases. In general, it appears that the majority of our eviscerated cases had a stormy postoperative course, complicated most frequently by increased abdominal pressure resulting from distention, vomiting, hiccough, and cough.

Type of Incision. Colp states that the incidence of evisceration is fundamentally dependent upon the effects of the underlying disease rather than the incision per se. The incision is only incidental to the approach to the underlying pathology. In our series evisceration followed upper abdominal incisions in nineteen cases, lower abdominal incisions in fifteen cases. Evisceration has followed every type of incision and closure. Maes, Boyce and McFetridge reported six cases following the McBurney incision and one evisceration following operation for direct hernia. In the 1,294 cases compiled by Jenkins, 474 followed upper abdominal incisions and 450 followed lower abdominal incisions.

Anesthesia. It is quite evident that the type of anesthetic per se plays an insignificant part, but the *character* of the anesthesia—the degrees of relaxation, which permits adequate exposure and facilitates handling of viscera with minimum of trauma, and most important, which permits a closure of the abdominal wall without tension—is significant. Spinal has

therefore been advocated because of the complete relaxation of the abdominal wall and intestines. Two of our patients had spinal, while practically all the rest had general ether anesthesia. The surgeon hesitates to incise the peritoneum with the patient rigid and straining, and he should be equally if not more hesitant to suture the peritoneum unless relaxation permits approximation of the peritoneum and posterior sheath without tension.

Closure. Layer closure was used in suturing the primary incision in all of our series, and in eight cases retention sutures were added: generally plain 2 continuous for peritoneum, chromic 2 interrupted for fascia, and silk or dermol for skin. From the literature, it appears that rupture apparently occurs quite as often with retention sutures as without them, which is not surprising since they do not enter the peritoneum—and the peritoneum is the layer in which rupture occurs first. Tension sutures tied too loosely give little support to the wound, and if tied too tightly cut through all the layers to some degree. Jenkins advises against using tension sutures in the presence of continuous catgut sutures, both because of the mechanical damage done to the continuous suture, and because of bacterial contamination extending along the tract from its introduction in the skin to its contact with the continuous catgut suture. Lahey,[39] Eliason[13] and Colp,[10] have eliminated the use of strangulating through-and-through stay sutures with a decrease in the liquefactive necroses, stitch abscesses, and definite improvement in wound healing.

In contrast, Kennedy,[32] reporting the combined surgical experience of himself and his predecessor, Dr. Joseph Price, during fifty-six years, states that during this time not a single evisceration occurred with the use of through-and-through silkworm sutures. Baldwin[2] makes the impressive statement that he performed 16,465 laparotomies using 2 chromic catgut throughout, without an evisceration. Howes and Harvey[29] have shown that the use of mat-

tress catgut sutures to close the fascia only increases the strength of the suture from 10 to 20 per cent, and does not warrant the increased amount of catgut.

Infection. In our series gross infection was present in only four cases. It must be granted that the characteristic case of evisceration is not associated with gross infection, though it must likewise be granted, as Howes and Harvey pointed out, that in the presence of infection, catgut rapidly loses its strength and is quickly absorbed regardless of chromicization.

When the infection is of low grade or of the nonsuppurative type it may be overlooked. The rapid digestion of chromic gut which has been frequently observed in apparently clean eviscerated wounds can be attributed to this low grade infection (Jenkins[30]). In the 1,294 cases reviewed by Jenkins an obvious infection of the wound was noted in 22 per cent of the eviscerated cases in which the condition of the wound was given.

Farr[16] relates an interesting sidelight on disrupted wounds reported at the Cornell Division of the New York Hospital during Dr. Gibson's tenure of office, viz., that in many hundreds of severely infected right rectus wounds with extensive peritonitis or abscess, the wounds were left wide open and packed with the Gibson-Mikulicz tampons. No sutures were used—and eviscerations were very rare. Technically, these wounds were all disrupted and practically, the patients recovered.

Drainage. Only six cases in our series required drainage. Theoretically, the institution of drainage should provide a ready-made exit for the protrusion of abdominal contents were it not for the fact that the organized adhesions which form snugly about the drain seal the space about it. In Colp's series the incidence of evisceration in drained cases was 1.22 per cent as compared with 0.84 per cent in undrained cases. Meleney and Howes' series of fifty cases showed a higher incidence of eviscerations in undrained than drained cases. It must be remembered, however, that the

TABLE IV
INCIDENCE OF DISRUPTIONS AND EVISCERATIONS IN RELATION TO DRAINAGE

Author	No. of Incisions	Drained	Incidence of Disruptions	Not Drained	Incidence of Disruptions
Colp......	2,750	1,147	13 (1.22 per cent)	1,603	13 (0.81 per cent)
Singleton...	9,000	1,816	18 (0.99 per cent)	7,184	43 (0.59 per cent)

total number of drained abdominal wounds is very much lower than those not drained.

In Jenkin's series of 1,294 American and European eviscerations, 153, or 17 per cent, were drained. In the American series a third of the reported cases were drained. Jenkins reminds us that "the use of a drain usually implies the presence of infection and therefore the presence of the intra-peritoneal infection is of more significance as an etiological factor than the drain itself."

Since catgut is rapidly digested in the presence of infection, the tensile strength and continuity of a continuous suture in contact with the draining wound would be destroyed long before adequate healing had occurred in the rest of the wound. Jenkins suggests therefore that when feasible a separate stab incision be made for drainage or interrupted sutures be used for closure. Even when a drain is used as a precautionary measure as in cholecystectomy, he draws our attention to the possibility that the drain may permit bacterial contamination from the skin to the depth of the wound which may hasten catgut digestion.

Day of Rupture. It is interesting to note the frequency with which evisceration is recorded immediately following removal of skin sutures. In our series twenty-seven of the thirty-four eviscerations occurred between the sixth and seventh day. The greatest number occurred on the sixth postoperative day. One case (cecostomy) eviscerated one

foot of the colon one day postoperative. Meleney and Howes report thirty-two of their fifty cases occurring between the seventh and tenth day. But disruption can occur at other times. Colp, Grace, Meleney, and Howes, report the accident on the second and third postoperative days. In thirteen of the cases collected by Sokolov, it occurred after the twentieth day, in one instance as late as the twelfth year postoperatively. In seven of Maes, Boyce and McFetridge's series of forty-four cases it occurred after the fifteenth day. Three of their patients who had been discharged from the hospital returned on the third, fourth and the seventy-fifth day following discharge with ruptured wounds. In Jenkin's series of 1,294 collected cases the average time of evisceration was 8.3 days postoperatively.

Does evisceration actually occur then? Is evisceration a suddenly developed complication? It is a common belief that the evisceration of a wound is a process that occurs simultaneously throughout the wall. To the contrary, evisceration is usually a process that occurs slowly and in steps. Ries[55] has pointed out the following facts: "Examination of the wound edges immediately after evisceration will show signs of bleeding in the skin edges but nowhere else. The peritoneum, muscle and fascial layers will be edematous, matted together with the appearance of chronic inflammation and no evidence of recent injury."

It is my belief that eviscerations are erroneously being recorded as occurring most frequently between the sixth and tenth day which coincides with the usual time of removal of skin sutures. I believe that when the skin sutures are removed the underlying eviscerated bowel is born to view and that the evisceration was completed days previously as evidenced by the adhesions and fibrous exudate covering the intestines, the omentum and peritoneum.

Eviscerated Contents. The small intestines and omentum were most fre-quently found as the eviscerated contents in our series. In three instances the large bowel eviscerated. In one case the stomach and small intestines, and in one case the liver, stomach and small intestine were eviscerated.

TABLE V
ANALYSIS OF DATA ON THIRTY-FOUR EVISCERATED CASES
Age. 40–50
Sex
    Male—20
    Female—14
Deaths—17
Mortality—50 per cent .
Primary disease
    Carcinoma found in 12 cases (35.2 per cent)
        Generalized—1
        Pancreas—1
        Stomach—5
        Intestine—4
        Ovary—1
    Gall-bladder disease—4
    Gyn. and Obst.—6
    Appendicitis—3
    Stomach and duodenum—7
    Spleen—1
    Ventral hernia—1
Excision of previous scars—5
Preoperative condition
    Good—12
    Poor—18
Postoperative course
    Respiratory infections—9
    Persistent vomiting and distention—13
    Undue exertion—4
Incisions
    Upper abdominal—19
    Lower abdominal—15
Anesthesia
    General—32
    Spinal—2
Infection—4
Drainage—6
Closure
    Layer—26
    Layer plus retentions—8
Treatment. Through-and through silkworm—21
    Secondary suture—32. Through-and through plus layer—6
    Layer closure—5
    Adhesive strapping—2

DIAGNOSIS

A majority of cases of disruption are not recognized or even suspected until the skin gives way and a viscus protrudes.

Separation of the deeper layers of an incision may often be diagnosed by the presence of a slight fulness or bulging of the wound during the postoperative

period, even though the skin appears perfectly healed. Careful separation of the skin edges discloses the presence of omentum immediately beneath the skin. More frequently, however, one's attention is called to this complication by the *presence of a serosanguinous discharge.* Totten[63] reported that serosanguinous discharge preceded evisceration in 44 per cent of his forty-seven cases. Less frequently the patient may suddenly experience severe pain in the operative site while straining, retching or vomiting, and complete rupture may occur suddenly without warning.

Clinically these patients often continue to have abdominal discomfort after the third postoperative day. Distention after repeated enemas, hiccough and belching is frequent. Symptoms suggestive of partial intestinal obstruction predominate. Such a symptom complex should always lead one to inspect the wound for possible evisceration.

TREATMENT

The two chief methods of dealing with an evisceration are packing and secondary suture.

Tampon treatment is a method of choice in infected cases, and in those desperately ill in whom evisceration has occurred secondary to generalized peritonitis. It consists of gently replacing the contents within the abdomen and packing the gaping wound snugly with plain or iodoform gauze. The wound edges are approximated with a few adhesive strips. Healing requires about thirty-seven days as a rule. Colp reports that in nineteen cases treated by tampon, eleven developed hernia. This is a serious objection to tampon treatment.

Prompt secondary suture of the abdominal incision has been recommended as the most satisfactory method of treatment. Heavy silk, silkworm gut, and silver wire are the suture materials most commonly used. The sutures are usually interrupted and placed through-and-through all layers of the abdominal wall. Lahey,

Starr and Nason, state that peritoneal infections following wound rupture is rare in their experience and for this reason advocate closing the wound without drainage.

In our series of thirty-four cases, twenty-one had through-and-through silkworm suture; six cases had through-and-through plus layer closure; five cases had layer closure only; two had no suture but were strapped with adhesive.

Of the seventeen patients who recovered following secondary suture, thirteen were followed up—and five of the thirteen developed ventral herniae.

ETIOLOGY AND MECHANISM

Colp[10] believes that the primary disease is the most important factor underlying the etiology of evisceration. While it is true that evisceration may occur in any disease affecting the peritoneum or its contents—it is definitely associated more frequently with some lesions than with others. Our series has shown a definite relation between incidence of evisceration and carcinoma. It would seem logical to suppose that illnesses of a protracted nature attended by emaciation, anemia, cachexia, might devitalize the patient sufficiently to interfere with reparative powers and wound healing. Yet if we are to conclude that the recuperative power of the patient is a factor in the causation of evisceration it is difficult to explain the paradoxical condition of *failure of the primary suture* and *success of the secondary closure.* Grace[20] reported that of twenty-eight cases that required secondary suture, in only two cases did the wound fail to heal and a rerupture occur.

Erdmann[14] ascribed disruption of wounds to "tissue hunger" with resulting rapid digestion and absorption of catgut. Clute,[9] Meleney and Howes[45] believe that the solution of the problem lies in the attention paid to the actual direct suturing of the peritoneum and posterior sheath. Harvey[23] ascribes the three essential reasons for disruption of wounds to: (1) failure properly to close the posterior

rectus sheath; (2) the accumulation of serum in dead spaces; and (3) the use of catgut of too large a size and in too large a quantity, which increases the exudative reaction and delays the onset of fibroplasia.

intra-abdominal pressure as the underlying factor in the etiology of eviscerations.

Thompson,[62] Ravdin[53] and Whipple[65] have noted that deficiency of blood proteins retards healing and enhances wound

TABLE VI
SUMMARY OF DATA ON 621 AMERICAN WOUND DISRUPTIONS AND EVISCERATIONS

	No. Cases	Average Age	Per Cent Male	Per Cent Female	Per Cent Malignant	Per Cent Biliary Tract	Per Cent Stomach and Duodenum	Per Cent Gyn. and Obst.	Per Cent Appendix	Per Cent Mortality	Per Cent Infection	Per Cent Upper Abdominal Incision	Per Cent Lower Abdominal Incision	Per Cent Drained
Bowen..............	34	45	59	41	35.2	11.76	20.6	17.6	9	50	12	56	44	18
Fallis...............	50	45	55	45	16.3	20.4	14.3	28.6	16.3	34	20	53	47	38.5
Glenn and Moore......	22	50	91	9	27	27	13.5	0	3.5	45.5	27.2	68	32	36.3
Hinton..............	19	42	70	30	10.5	37	60	0	0	21	16	95	5	21
Singleton and Blocker...	61	36	27	73	6.5	10	3.25	65.5	11.5	31	60.7	15	85	29.5
Totten..............	47	45	80	20	15	7.2	15	8.5	6.4	40	36	51	49	
Total and average......	232	44	63.6	36.4	18.4	18.86	21	20.3	7.8	36.9	28.6	56.3	43.7	28.6
Jenkins—Review of 389 American cases........	389	46	63	37	26	19	14	12	12	39	34	59	41	31
Total 621 American cases and average percentage	621	44.5	63.3	36.7	22.2	18.9	17.5	16.1	9.9	37.9	31	57.6	42.3	29.8

Kraissl,[37] Hinton,[26] and Babcock[1] have shown that allergic reactions to catgut proteins and sensitivity to chemicals used in processing chromic gut may be factors responsible for more rapid digestions of catgut in some patients. Kraissl carried out a controlled series of experiments in guinea pigs previously sensitized to catgut and reported that 35.7 per cent disrupted their abdominal wounds; 32.1 per cent healed abnormally, and 32.1 per cent healed normally. He also sensitized guinea pigs to chromic acid and 25 per cent disrupted; 37.5 per cent had abnormal healing and 37.5 per cent healed normally.

If we recall that catgut is a foreign protein it seems logical to assume that a certain percentage of patients might be considered allergic to it.

Jenkins,[30] Clute,[9] Meleney and Howes[45] stress the importance of adequate closure of the posterior rectus sheath in minimizing the incidence of evisceration. Milbert[46] and Glasser[18] emphasize the rôle of increased

disruption. Whipple states that diminished serum proteins of the blood and tissue and vitamin deficiencies prevent normal healing in many of the elderly patients. Wallboa (1926) demonstrated that wounds in animals with scurvy will not heal. Lund at Boston City Hospital finds a great number of patients with miscellaneous surgical conditions also run suboptimal levels of vitamin c—many of the levels approximately close to scurvy.

If other biochemical factors are responsible for the delayed wound healing the nature of them are at present unknown.

The most reasonable theory yet advanced as for the mechanism of evisceration is that of Freeman.[17] His idea is that through an opening in the peritoneal layer which may be present as a result of inadequate closure or develop as a result of increased intra-abdominal pressure due to distention, vomiting, hiccoughing, coughing—the sutures tear through the peritoneal layer, a wedge of omentum

protrudes early postoperatively and the piston-like action of the omental wedge forcibly and intermittently thrust by the respiratory excursions, vomiting, coughing, and hiccoughing, makes the opening larger and larger, permitting not only omentum but intestines to protrude. The theory is plausible because of: (1) the usual tendency of the omentum to enter and fill the wound cavity when primary closure is undertaken; (2) omentum is practically always present in eviscerated wounds; (3) the swollen state and the associated adhesions suggest that the prolapse has occurred sometime previously rather than at the time of suture removal; (4) the incarceration of the omentum and intestines is manifested clinically by the symptoms of obstruction which frequently appear very soon after operation.

Suture Material.   Since we are relying on the protein of a sheep's intestine to maintain approximation of incised tissues, it behooves us to acquaint ourselves with the extrinsic and intrinsic factors in manufactured catgut which may affect the tissues locally and generally—influencing absorption, digestion, tissue reaction, and allergy.

Catgut is made from twisted ribbons of the submucosa of sheeps' intestines. It is a collagen which is markedly hygroscopic, and when brought into contact with water, it rapidly absorbs moisture, softens, and swells. Boilable catgut is completely dehydrated and kept in tubing fluid of anhydrous toluol. Consequently, boilable catgut is stiff, wiry, inflexible, and must be moistened to restore its pliability. Prolonged immersion in water before use causes considerable swelling of the collagen, and decreases its tensile strength. Soaking boilable catgut in hot water is dangerous, since it hydrolizes the collagen and destroys its tensile strength (Davis and Geck[11]).

To date there are no tests that can determine whether a given brand of catgut will or will not resist absorption for a sufficient length of time for it to be reliable for all requirements of suture material. In numerous cases of this series the operators on resuture reported the complete digestion of catgut after the third to fifth day. While the question of absorbability of catgut is of great importance, yet the committee of catgut standards of the American Medical Association has not been able to persuade catgut firms to agree on any standards to which they can or will make their products conform.

Because boilable gut is completely dehydrated it absorbs more of the digestive media than the non-boilable variety, and according to Kraissl[36] non-boilable gut may be depended to remain in the tissues longer than the boilable catgut.

Non-boilable gut is basically prepared the same as the boilable, but placed in tubing fluid of 95 per cent alcohol and 5 per cent water. A smooth flexible gut is produced which requires no soaking before use.

It is not considered good technique for the surgical nurse to test the tensile strength of the suture before handing it over to the surgeon. A few sudden pulls or jerks are similar to the method used in breaking the cord after tying a parcel, and may so weaken the suture as to permit it to break under slight tension when in use.

The customary terms "ten day," "twenty day" catgut are used to indicate the absorption time of the suture when embedded in normal muscular tissues of aseptic wounds. The manufacturers[11,31,40] specifically state that in mucous and serous membranes it will last from *one-third to one-half* of this period. It is paradoxical therefore that it has been customary to use plain for peritoneum and chromic for fascia. Since the peritoneum is a serous membrane it is suggested that No. o or No. 1 forty day chromic gut be used for peritoneal closure.

In suturing other serous covered viscera we are meticulously careful to use atraumatic needles. Yet, frequently in suturing

the thin, friable, single mesolethial layers of peritoneum, large bayonet-shaped needles carrying two thicknesses of catgut stab the peritoneum, and wound and puncture it unnecessarily. The writer suggests the use of the curved atraumatic needles carrying o or 1 forty day chromic for peritoneal suture.

There is no point in introducing a suture into a tissue which has a greater tensile strength than the tissue itself (Harvey).[23] Catgut is a foreign protein and is absorbed by an exudative reaction, which, when excessive delays wound healing. To diminish the exudative reaction it is most advisable to bury the least amount of catgut within the tissues. Harvey reminds us that we have an exaggerated idea of the holding power of fascia in respect to the size of catgut used. He states that No. 1 or No. 2 chromic gut is about *ten times* stronger than the holding power of fascia itself. Harvey believes it is ridiculous to use o plain for approximation of fat tissue since fat itself has no holding power. The finest possible plain suture (No. 000) is more than sufficient to approximate fat. Howes[28] reminds us that the tensile strength of o chromic gut is greater than the holding power of any tissue likely to be sutured with it.

The most graphic demonstration of tissue reaction, catgut absorption, and healing with respect to size of catgut used was given by Bower[5] at the 1938 convention of the American Medical Association. Transparencies showing the results of sutures of stomach and intestine and photomicrographs showing tissue reaction and absorption rate of one and five zero chromic were demonstrated. There was a most marked local reaction; tissue union was delayed; absorption more rapid in using the coarser gut. The very fine five zero gut lasted longer, caused a minimum of local tissue reaction, more rapid healing and more complete healing than No. 1 chromic. One left the exhibit with a determination to use smaller sizes of catgut in the suture of tissues generally.

Recently experiments were reported in the attempt to adopt other animal proteins for suture materials. Bost[4] originally prepared "carnofil" a suture material made from the muscle tissue of the horse. It is said to be relatively free from bacteria and can be subjected to unlimited sterilization.

Preobrazensky[51] has used dogs' nerves removed aseptically, treated with 20 per cent acetic acid, twisted and sterilized, for intestinal sutures. Sidelnikoff[57] suggested the use of sutures made from the umbilical cord.

The chemical treatment of catgut for sterilization and control of absorption time merits study and consideration.

Clock[7] has detected non-sterility of 62½ per cent of twenty-four foreign brands of catgut and 50 per cent of twelve American brands. Clock came to the conclusion that chemical sterilization of catgut is inefficient and unreliable when subjected to careful bacteriologic examination and that controlled heat sterilization is the only positive method of sterilization of catgut sutures. Moreover Clock remarks that the embedding of metallically impregnated catgut frequently causes irritant action on the tissues—with excessive cellular activity and consequent hastened digestion and absorption of the catgut. Sensitivity to chromic acid has been reported by Kraissl.[37]

Reduction of the amount of catgut under the subcutaneous tissue has been the most important factor in reducing the incidence of serum collections.

The essentials of good suturing include the use of small size catgut. The advantages of using small sizes of catgut sutures are (Davis and Geck[11]):

1. They cause less trauma to the tissue at insertion than do coarser sutures.

2. They permit smaller knots, with less foreign protein.

3. They permit better approximation of tissues.

4. They decrease the sum total amount of foreign proteins; result in a minimum of

exudative reaction; favor more rapid and complete wound healing.

Absorption time may be altered by tanning, chromicizing, treatment with formalin, iodine, impregnating with mercury, silver and copper. The most resistant catgut is produced in foreign countries where it is frequently hardened with formalin. Kraissl[35] found that foreign gut hardened with formalin remained unchanged after three weeks. In Germany, Haefen[21] reported that iodized catgut absorbed very slowly, and remained in the tissues as long as four months.

The larger sizes of catgut do not necessarily last longer than the smaller sizes. The digestive enzymes penetrate between the twisted ribbons and digest each separately. In chromic gut, the chromicizing may be on the outer layer only, and when this is penetrated, digestion is rapid; while in the smaller sizes the chromicizing extends through the entire strand (Kraissl[36]).

In the presence of infection catgut is absorbed with the utmost rapidity. When catgut sutures are embedded in the tissues of persons allergic to catgut, premature absorption may occur.

Absorption time varies according to the nature of the tissues in which the sutures are imbedded. A well known manufacturer[40] of iodized tanned catgut states that the suture is more quickly absorbed in mucous and serous membranes—lasting from *one-third* to *one-half* the time it would if buried in muscle. Thus, a tanned "twenty" day suture would last only seven to ten days when buried in the peritoneum—since the peritoneum is a serous membrane.

In the repair of a lacerated perineum or cervix, any catgut, whether chromicized or not, will be digested and absorbed with greater rapidity than when buried in aseptic wounds, because of the digestive action of the enzymes in the cervical and vaginal secretions. Therefore, forty day chromic catgut is preferred for repair of the perineum, cervix and vagina. Hess[24] remarks that it should never be

necessary to use gut larger than No. 1 chromic, and reports the routine use of No. 00 chromic interrupted sutures in approximating episiotomy incisions and perineal lacerations.

Another well known manufacturer[11] states that No. 1 plain catgut loses its tensile strength in four and one-half days; No. 2 plain in five days. For practical purposes, a suture whose tensile strength is lost, can be considered as absorbed, although as a rule, complete absorption occurs about three days after complete loss of tensile strength.[11] Jenkins[30] conducted clinical tests on the duration of tensile strength of catgut and concluded that "plain catgut lasts five to six days by tension suture tests, but that it becomes untied in the tissues in two to three days."

Five days is too short a time to hold the peritoneum during the postoperative vomiting, distention, and respiratory complications with its accompanying increase in intra-abdominal pressure and tension on the peritoneal suture line. Since chromicized gut maintains its tensile strength regardless of size, the writer again urges the use of No. 1 forty day chromic for peritoneal closure.

During the past few years there has been a noticeable trend in the literature to rely more on non-absorbable material for sutures—particularly silk and metal alloys. Meleney,[45] Howes,[28] and Whipple,[65] believe that silk is a better suture material than catgut in clean cases. Maes, Boyce and McFetridge[43] remark that catgut used with the silk technique is probably as safe as silk. Silk should not be used in infected cases.

The use of metal wire appears to be gaining favor with surgeons in various parts of the world. Reid, Zinniger and Merrell[54] reported brilliant results in the use of silver wire. Babcock[1] has recommended the use of "Nobel" metal—an alloy of stainless steel wire. In France, Minine[47] employs an alloy called "nickeline" in gynecological operations.

Wound Healing. From experimental studies, Meleney and Howes[45] found that "the healing of a wound is disturbed only by profound systemic changes, but locally, healing is readily delayed by a retardation of the phase of fibroplasia as a result of excessive exudative reaction." Any factor which increases the exudative reaction delays wound healing. Infection, trauma, the presence of dead tissue such as blood or suture material act for the most part by retarding the onset of fibroplasia, rather than by inhibiting the growth of fibroblasts once initiated. These observations emphasize again the necessity for gentle handling of tissues, perfect hemostasis, and burying the least amount of suture material to decrease the exudation and hasten the onset of fibroplasia.

During the first or exudative phase of wound healing, the tissues are dependent for its approximation upon the suture material. In the second, or stage of fibroplasia, the wound begins to develop its own holding power. During the period when the wound itself is weakest, the sutures are strongest; and when the sutures begin to lose their strength the wound becomes increasingly strong.

Ogilvie[49] states "that a well sutured wound has a tensile strength of about 40 per cent of that of the tissues before they were cut. During the first few days the tissues undergo autolysis, while the sutures are being digested. By the fourth day the strength of the wound has dropped to some 20 to 25 per cent of the original. From the fifth day onward fibroblasts are laid down rapidly, and the strength increases; so that by the sixth day it is 50 per cent; and by the tenth day about 90 per cent of the normal."

Catgut is absorbed by an exudative reaction an excess of which retards fibroplasia and delays wound healing. *Bury the least amount of catgut to promote and hasten wound repair.*

The tragedy of evisceration is due not to the separation of the wound layers per se—but rather to the *intestinal obstruction* which results from the kinking and knuckling of loops of intestine which found their way through an opening in the peritoneum and became adherent to muscle or fascia. If the obstruction is relieved early enough —the patient recovers, whether we suture the wound or pack it with gauze. *Intestinal obstruction kills the patient—not the separation of the tissue layers.*

How many patients have separation of the deeper abdominal layers without obstruction of intestinal loops—or which develop incisional herniae, it is difficult to state. A majority of the patients with incisional hernias are unaware that they were on the borderline of eviscerations.

Neuhof[48] remarks that "postoperative incisional hernia may well be referable, in part at least to rupture with evisceration whose results may not be noted until months after operation."

King[33] in a study of 150 cases of incisional hernias found that 40 per cent of the hernial develop within a month after surgery. *Incisional hernia is frequently a delayed evisceration.* In many cases the abdominal contents lie directly in the superficial fascia or under the incisional scar. The symptoms are mild or severe depending upon the dragging and kinking of the adherent visceral contents.

Were it possible to maintain the integrity of the peritoneal suture line during the postoperative period—then even if all other layers gave way—there would be an absence of the morbid intestinal obstruction which kills the patient. The intact peritoneum is able to withstand increased intra-abdominal pressure.

As surgeons we perform this experiment in every laparotomy. We incise all layers down to peritoneum—yet even when the patient is having a stormy anesthesia— straining, coughing—we observe the peritoneal layer flap as a sail in the wind with changes in the intra-abdominal pressure. But once the peritoneum is incised, it frequently takes four hands and as many sponges to retain the viscera within the abdomen.

It is my humble opinion that if we were able to approximate carefully the incised

peritoneum with a suture which would remain long enough to tide the patient over the postoperative period—and if it were possible to maintain the equilibrium of that peritoneal suture line long enough to permit healing by minimizing those factors which produce increased intra-abdominal pressure; and utilize incisions, approaches, and closures which support the peritoneum from without—then we should be decreasing the incidence and mortality of eviscerations. As long as the omentum and intestinal coils slide along peritonealized surfaces the incidence of obstruction is minimized. Large scrotal and ventral hernias can hold a considerable volume of intestinal contents within their sacs—and only occasionally become obstructed. But once the omentum or intestine leaves the peritoneal cavity it becomes adherent, kinked, and obstructed.

In stressing the importance of the peritoneal layer, I do not underestimate the rôle of increased intra-abdominal pressure, but rather emphasize the effect of increased intra-abdominal tension against a peritoneal incision which has been inadequately sutured, or approximated by a suture which was too rapidly absorbed in cases complicated by a stormy convalescence.

PREVENTION OF EVISCERATION

Let us proceed to consider logically the measures suggested and advocated in preventing and minimizing the incidence of evisceration:

1. Preoperative preparation of cachectic, dehydrated patients by transfusions and intravenous fluids to improve their general condition, supply serum pro eins and blood volume.

2. Anesthesia which will give sufficient relaxation, permit exploration with a minimum of handling of the viscera, and permit closure of the peritoneum without tension.

3. An incision which will give the most direct approach to the pathology with a minimum of traction and trauma—the closure and approximation of which is favored by the position which the patient is to assume.

4. The protection of tissues from mechanical and chemical damage; clean cut dissections; the avoidance of mass ligations; perfect hemostasis.

5. The meticulous closure of the peritoneal layer together with the posterior rectus sheath with fine forty day chromic catgut on an atraumatic needle.

6. Burying the least amount of catgut in the tissues to minimize exudative reaction—using No. 1 forty day chromic for peritoneum; No. 0 chromic for fascia and muscle, and 000 plain for subcutaneous suture.

7. The use of through-and-through non-absorbable sutures alone or in combination with layer closure in cancer, gallbladder and stomach cases.

8. The prevention and suppression of postoperative restlessness and undue activity.

9. The early use of the Levine catheter, Connell suction, in the treatment of gastric dilatation, distention and vomiting.

10. Change of posture, CO_2 inhalations to decrease hypostatic pulmonary congestion and atelectasis.

11. Prompt investigation of persistent vomiting by inspection of the wound—probing if necessary to determine the possible presence of deep separation of the layers.

12. Immediate inspection of the wound when the patient reports "something having given way."

13. Recognition of a serosanguinous discharge as an important warning sign of the presence of an evisceration.

14. The prompt secondary closure of the eviscerated wound.

In the attempt to reduce the incidence of eviscerations especially in those cases where the incidence is known to be great, e.g., in carcinoma, stomach and gallbladder cases, surgeons have adopted various techniques in closure. Colp[10] advocates the use of interrupted heavy through-and-through braided silk sutures in closing median epigastric incisions made

especially for gastroduodenal ulcers and practically for all carcinomas of the stomach. White,[66] noting the success with which firm union was obtained on secondary closures with through-and-through

transversely, there is considerable difficulty in closure and frequent failure of holding in this layer. Clute,[9] Jenkins,[30] Pool,[50] Meleney and Howes[45] considered failure of this layer to hold as an important

TABLE VII

SUMMARY OF DATA ON 1,526 CASES OF WOUND DISRUPTIONS AND EVISCERATIONS
(905 European and 621 American Cases)

	No. Cases	Average Age	Per Cent Male	Per Cent Female	Per Cent Malignant	Per Cent Biliary Tract	Per Cent Stomach and Duodenum	Per Cent Gyn. and Obst.	Per Cent Appendix	Per Cent Mortality	Per Cent Infection	Per Cent Upper Abdominal Incision	Per Cent Lower Abdominal Incision	Per Cent Drained
Jenkins—Summary of 389 American and 905 European cases.	1249	44	57	43	25	12	15	18	15	35	22	51	49	17
Bowen—Summary of 232 American cases .	232	44	63.3	36.4	18.4	18.86	21	20.3	7.8	36.9	28.6	56.3	43.7	28.6
Total and average percentage	1526	44	60.3	39.7	21.7	19.43	18	19	11.4	35.9	25.3	53.6	46.4	28.2

sutures, was induced to adopt the same procedure with fresh wounds of suspected cases. Kennedy[32] uses no absorbable sutures in his wounds. The combined surgical

TABLE VIII

INCIDENCE OF DISRUPTIONS AND HERNIA IN VERTICAL AND TRANSVERSE INCISIONS (SINGLETON)[58]

Incision	No.	Disruptions	Hernia
Vertical	292	9 (3.08 per cent)	16 (5.47 per cent)
Transverse....	470	0	1 (0.21 per cent)

experience of himself and his predecessor, Dr. Joseph Price, during fifty-six years, contained not a single case of evisceration with the use of through-and-through silkworm gut sutures. Baldwin[2] performed 16,465 laparotomies using No. 2 chromic throughout and silkworm gut for stay sutures without an evisceration. Mont Reid[54] has used a silver wire technique and reports that not a single evisceration occurred in 334 cases in which it was anticipated.

Because of the anatomic structure of the posterior rectus sheath, above the semilunar line, with its fibers running

factor in the etiology of eviscerations. For the same reason, numerous operators, Sloan,[59] Mayo,[44] Singleton,[58] Whipple,[65] have adopted transverse incisions to approach upper abdominal pathology. A transverse incision permits approximation of the posterior sheath without tension, and the position which the patient assumes favors the closure of a transverse wound. Mayo advises the use of a longitudinal with a connecting horizontal incision for epigastric lesions.

Singleton, reviewing 9,000 consecutive abdominal operations at the John Sealy Hospital, presented facts to prove the disappearance of disruptions and postoperative herniae since the adoption of transverse approaches to both upper and lower abdominal procedures.

SUMMARY

1. Certain primary diseases, especially cancer and operations on the stomach and biliary tract, predispose to evisceration.

2. In a majority of the eviscerated cases, the preoperative condition was poor; and the postoperative course complicated by distention, vomiting and cough.

3. Evisceration has followed every type of incision and closure.

4. The tragedy of evisceration is due to its complicating intestinal obstruction.

5. The mortality in eviscerated cases is high—16 to 75 per cent.

6. Evisceration takes place in the first few postoperative days and is *born to view* usually at the time the skin sutures are removed.

7. A hiatus in the peritoneum due to inadequate closure; early absorption of catgut; increased intra-abdominal pressure, permit the intra-abdominal contents to escape.

8. To Jenkins[30] series of 389 American and 903 European cases, the author has added 232 additional American cases and tabulated the data on the total of 1,526 cases.

CONCLUSIONS

1. Careful preoperative preparation is necessary—transfusions in debilitated cases to restore blood proteins and volume.

2. Spinal or epidural anesthesia is used for relaxation to minimize handling of the viscera and avoid vigorous traction.

3. More frequent use of transverse incisions in upper abdominal approaches is advocated.

4. In carcinoma cases, gastric and biliary surgery layer closure is preferably reinforced by non-absorbable through-and-through sutures.

5. The posterior rectus sheath should be identified and included in the peritoneal suture in upper abdominal closures.

6. No. 1 forty day chromic is advocated for meticulous peritoneal closure, preferably on an atraumatic needle.

7. The equilibrium of the peritoneal suture line must be maintained long enough to permit healing by: (1) using a suture which will maintain its tensile strength for an adequate time; (2) recognition and immediate energetic treatment of complications, such as distention, vomiting, cough, and restlessness, which increase intra-abdominal pressure.

8. Prompt inspection of the wound should be made in persistent postoperative vomiting and the presence of a serosanguinous discharge should lead to a suspicion of evisceration.

9. The most common method of treatment of eviscerations is secondary closure with through-and-through non-absorbable sutures.

10. Acquaintance with the intrinsic and extrinsic factors in the varieties of manufactured catgut will lead to their more intelligent use.

11. A plea is made for the use of the finer sizes of catgut to enhance the *"art in sutures"*; to decrease the exudative reaction and hasten healing of abdominal wounds.

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THE SUPERIORITY OF VERY FINE CATGUT IN GASTROINTESTINAL SURGERY*

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FOR many years we have believed that the trauma induced by the passage of the usual large atraumatic needle with large sized catgut through delicate membranes is excessive. The objective of this research was to eliminate or reduce this trauma and tissue reaction, and to overcome some of the mechanical difficulties in securing accurate apposition and complete hemostasis of membranous tissue of the gastrointestinal tract. A reliable manufacturer† of catgut was asked to coöperate in our experimental investigations, and furnished us with the finest sized chromic catgut possible, affixed to appropriate sized atraumatic needles. The work described was started in April, 1937. So far as we have been able to determine, the technique employed in gastric resections and the comparative studies of the reactions of the tissues to the coarse and fine catgut have never before been done, nor have the experiments described in Series VI and VII of this paper.

METHODS AND MATERIALS

The dog was selected for the experiments because its stomach closely resembles that of man in size, shape and blood supply. While the total hydrochloric acid secreted in the dog's stomach is proportionately greater than in that of man, we concluded that any detrimental effect on the results could be discounted since both sizes of catgut would be exposed to the same degree of acidity.

† Davis & Geck, Inc.

Except in Series VI and VII, adult dogs weighing an average of 10 kg. were used. Food was withheld for twenty-four hours; at operation the abdomen was shaved and painted with 3.5 per cent tincture of iodine. An aseptic technique similar to that followed in operations on man was used. Under ether anesthesia, a vertical mid-epigastric incision was made, the stomach delivered, rubber faced clamps applied and the stomach divided vertically in the pars media. The blood supply was disturbed as little as possible, only small vessels being ligated.

In an attempt to determine the best method of suturing the mucous membrane, continuous sutures of the running type, over and over, and those placed so as to evert the mucous membrane, were used. In several instances in the anterior wall of the stomach the mucosa was approximated with a single continuous over and over stitch and the posterior wall with the Connell stitch. In our early series, we used three rows of continuous sutures in approximately 23 per cent of the operations; later, however, we used two rows, one to suture the serosa and one the mucous membrane. The abdominal wall was closed in layers with continuous silk sutures in the peritoneum, muscle, fascia and skin; the skin was painted with tincture of benzoin. The dogs were given water as soon as they reacted from the anesthetic and soft food at the end of thirty-six to forty-eight hours.

In studying the cross sections prepared for microscopic comparison, the tissue reactions to the different sizes of catgut in the

PLATE I

A

B

C

D
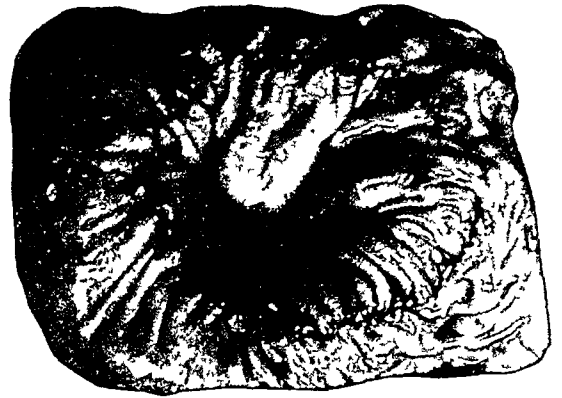
Gross specimens of adult dogs' stomachs, sutured following vertical division with 5-0 chromic and 0 chromic catgut.   Continuous over-and-over sutures removed at the end of twenty-four and 120 hours

A, twenty-four hours. 5-0 chromic. The edges of the mucous membrane were uniformly apposed. Because of the minimal necrosis of cells, the catgut, including the knot, was not exposed and edema was slight. The irregular discolored areas 0.5 to 1 cm. distant from the suture line were the result of operative trauma. These submucous capillary hemorrhages were usually absorbed within forty-eight hours.

B, twenty-four hours. 0 chromic. The edges of the mucous membrane were not uniformly apposed. The trauma associated with the passage of the large sized needle and catgut was responsible for cellular necrosis which, in turn, was responsible for the exposure of the catgut strand including the knot. Edema and ecchymosis were marked.

C, 120 hours. 5-0 chromic. Accurate primary apposition of mucosal edges; minimal destruction and subsequent absence of necrosis of cells and less tissue reaction account for the lack of scar tissue at the site of the incision.

D, 120 hours. 0 chromic. The necrosis of the cells in the mucosal edges followed by the exposure of the catgut strand and maximal tissue reaction account for the greater scar formation at the site of incision of the divided mucous membrane.*

* The use of a hand lens will enable the reader to see details more clearly and reveal the third dimension in the photographs.

stomach and intestine were found to be constant in the submucosa and the tissues beneath. In attempting to study the therefore, that the specimens would have to be prepared by a different method, one which, would show these reactions. This

Fig. 1. Twenty-four hours. A, 5-0 chromic—note slight degree of ecchymosis in the mucosa. Very slight edema close to the suture line; mucosal edges were invariably perfectly cemented together; necrosis minimal. B, 0 chromic—edge of mucosa ragged and necrotic; edema marked. Note partial exposure of suture line and untied knot. (See Plate 1, A and B.)

reaction of the cells in the mucous membrane, however, the cross sections were found to be unsatisfactory. It was evident, was accomplished by using a modified Spalteholz technique. Sections of dogs' stomachs and intestines were rendered

transparent after varying periods of time, following suture of the divided mucosa and serous layers.

needles of suitable sizes; and for some of the clinical investigations, small half-circle atraumatic needles were used.
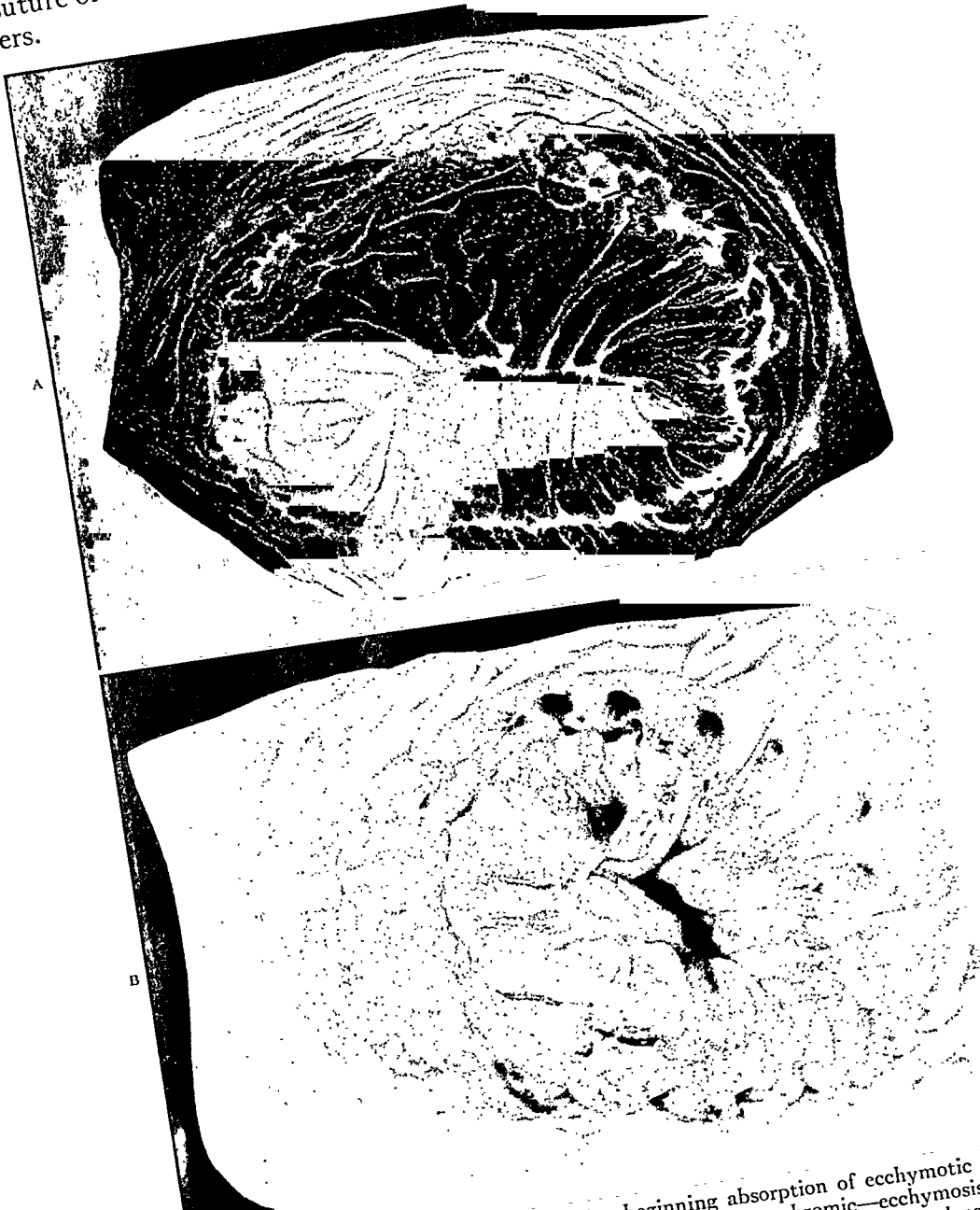
FIG. 2. Forty-eight hours. A, 5-0 chromic—beginning absorption of ecchymotic area; apposition of edges uniform; catgut not exposed. B, 0 chromic—ecchymosis and inflammatory edema slightly diminished; mucous membrane necrotic and covered with puroplastic exudate; note exposure of catgut.

Suture material comprised chromic catgut, sizes 0 and 5-0; and iodized catgut in corresponding sizes. The sutures were affixed to straight, taper point atraumatic

EXPERIMENTAL INVESTIGATIONS
SERIES I

Five pairs of dogs were operated upon. No. 0 chromic catgut was used to anasto-

mose one divided stomach, 5-0 chromic the other of each pair. The entire suture line with approximately 3 cm. of contiguous

No. 1 or No. 11, with Polya modification, was used to close the stomach.

At the time of removal, each specimen

FIG. 3. Seventy-two hours. A, 5-0 chromic—almost complete disappearance of ecchymosis; slight contraction of mucosal edges. B, o chromic—ecchymosis diminished; edema same as in forty-eight hour group. Necrosis increased; note exposure of catgut.

gastric wall on either side was removed in each case at a secondary operation, one pair at twenty-four, one at forty-eight, seventy-two, ninety-six and 120 hours. A Billroth

was placed so as to avoid tension in a position showing the suture line and a color photograph taken. It was placed in water for twelve hours, then in Kaiserling No. 1

solution for twenty-four hours, after which it was mounted and permanently placed in Kaiserling No. 2 solution.

twenty-four, forty-eight and seventy-two hour pairs, varying amounts of free sero-sanguinous fluid were encountered; in the

FIG. 4. Ninety-six hours. A, 5-0 chromic—complete disappearance of ecchymosis; further contraction of mucosal edges. B, 0 chromic—note exposed catgut and necrosis of mucosal edges.

Gross Appearance of Gastric Specimens. Without exception, reoperations at the end of twenty-four hours showed the sutured serous coats covered with plastic lymph; usually the omentum and less frequently the small intestine were cemented over the suture line on the anterior surface. In the

ninety-six and 120 hour pairs this fluid was usually not present. Unless great care was taken in separating the omentum and intestine to deliver the stomach for resection, the anastomosis was frequently disrupted where o chromic was used, particularly in the seventy-two and ninety-six

hour dogs. Microscopic inspection of the catgut and tissue changes readily explains this, the larger size catgut disintegrating

unaffected and little or no necrosis occurred in the surrounding tissues. (Figs. 1–5.)

Summary of Gross Changes of Suture of
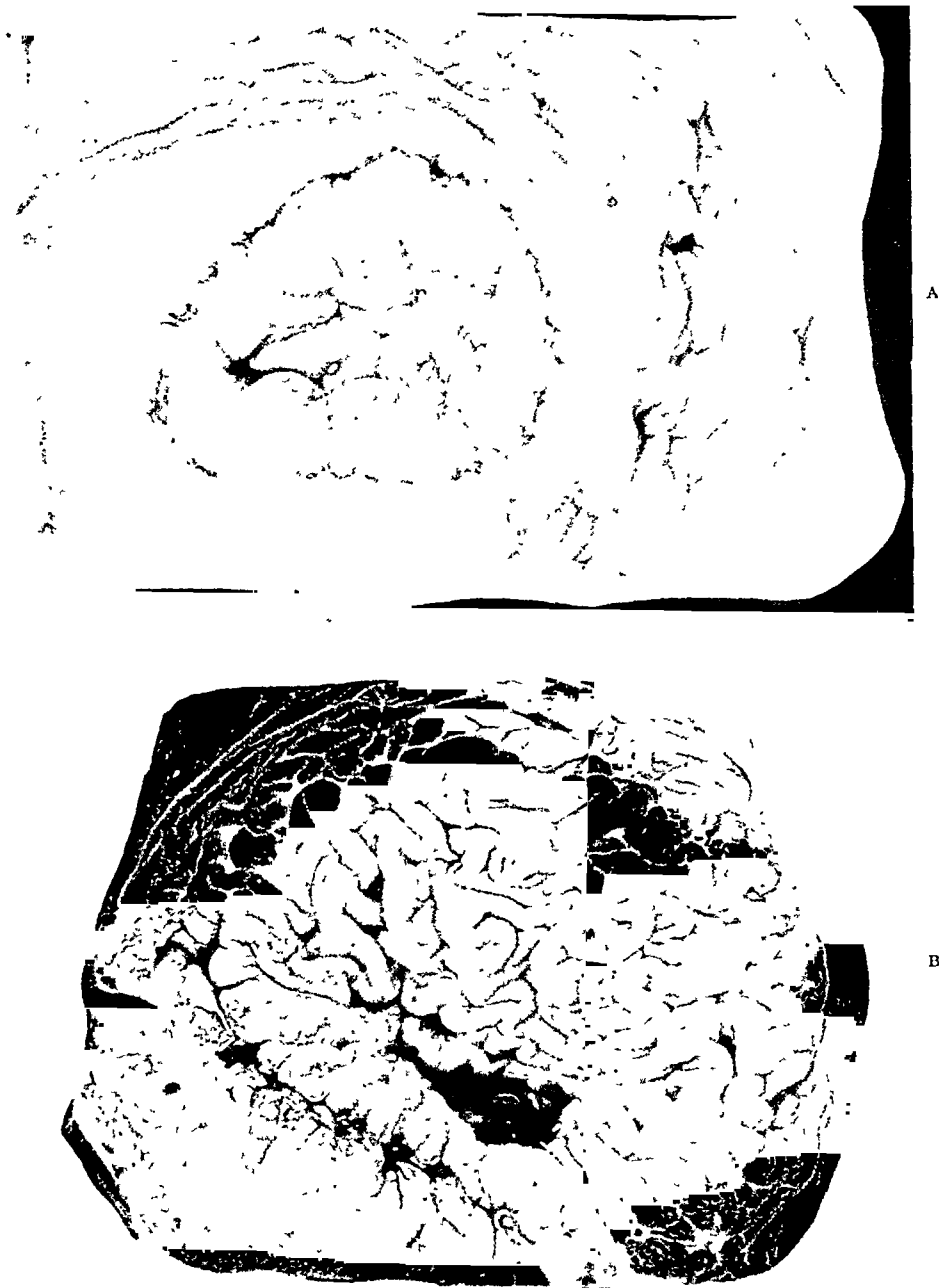
Fig. 5. One hundred and twenty hours. A, 5-0 chromic—accurate complete mucosal union. B, o chromic—union of mucous membrane incomplete in certain areas. Where complete, line of apposition distinctly wider than that following suture with the finer catgut. (See Plate 1, c and d.)

after seventy-two hours. Necrosis of the cells of the mucous membrane contiguous to the catgut was complete at 120 hours. The 5-0 chromic, however, was practically

Mucosa, Submucosa and Serosa of the Stomach of the Dog Following Vertical Bisection, Using 5-0 Chromic and o Chromic Catgut. The gross mucosal changes in the

specimens sutured with 5-0 chromic catgut showed immediate accurate primary union and a strong anastomotic line due to the

Figures 6 to 11 show the cross sections of tissue and catgut strand 5-0 chromic and 0 chromic. They demonstrate very clearly
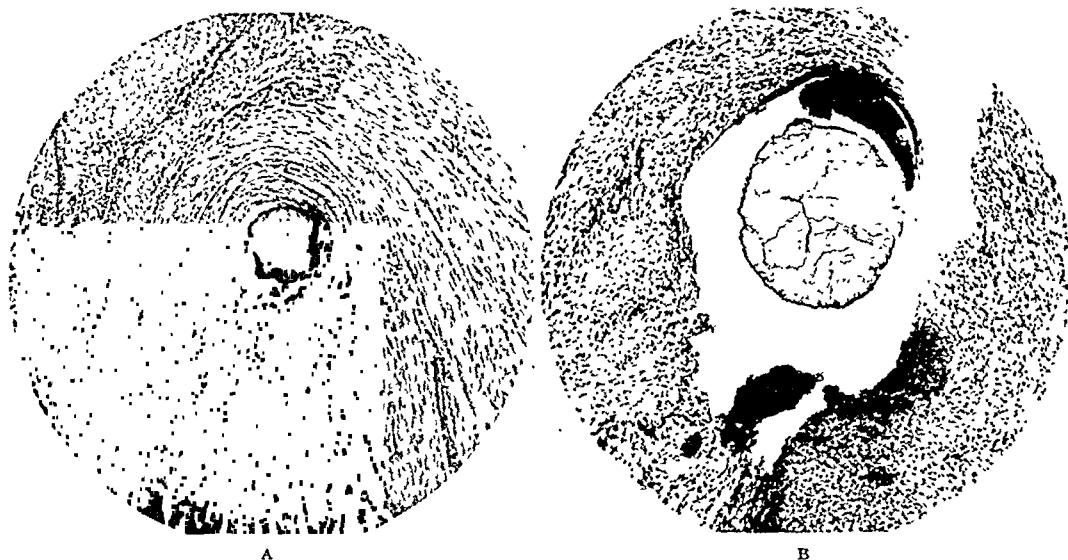
FIG. 6. Twenty-four hours. A, 5-0 chromic—mucosal openings small due to fine caliber of needle and minimum trauma associated with the drawing through of the catgut strand. Catgut is intact and leucocytic infiltration slight. B, 0 chromic—mucosal openings large due to the passage of a large caliber needle; marked trauma induced by the drawing through of the catgut strand. Catgut intact; marked leucocytic infiltration.

absence of necrosis. The gross mucosal changes in the specimens sutured with 0 chromic showed early necrosis of mucosal edges extending over a period of seventy-two to ninety-six hours, exposure of the catgut strand, frequent untying of the catgut knot, and a minimum of accurate primary union. (Plate I.)

SERIES II

Another five pairs of dogs were selected and the identical technique and operation of Series I carried out. The anastomosis with contiguous gastric mucosa was removed, as in the first series, one pair at each twenty-four-hour interval to 120 hours. After being photographed, they were placed in 10 per cent formalin and then prepared for microscopic examination. The paraffin technique was used and the sections stained with hematoxylin-eosin. An average of four to six sections were made from each anastomosis and, after being studied, those showing the maximum absorption of catgut were selected for photomicrographs.

the wide difference in absorption and tissue reaction between the two sizes of catgut.

Summary of Microscopic Changes. 5-0 *Chromic.* Minimal destruction of cells because of fine caliber of needle and catgut; minimal tissue irritation shown by almost complete absence of leukocytic infiltration; prolonged retention of catgut in the tissues.

0 *Chromic.* Maximum destruction of cells due to large caliber of needle and the drawing through of a large catgut strand; early absorption of catgut beginning at the end of seventy-two hours, more marked at the end of ninety-six hours and almost complete at the end of 120 hours. Leucocytic infiltration of the tissues accompanying absorption increased commensurately, reaching its maximum on the fifth day.

SERIES III

Gastroduodenostomies were performed on five pairs of dogs, using 5-0 chromicized and 0 iodized catgut. The serosa, muscularis and the mucosa were approximated with two rows of sutures; accurate apposition

was difficult because of the friability of the mucosa of the dog's duodenum. The preparation of the animals, operative tech-

5-0 *Chromicized.* The gross changes were identical with those observed in Series I.
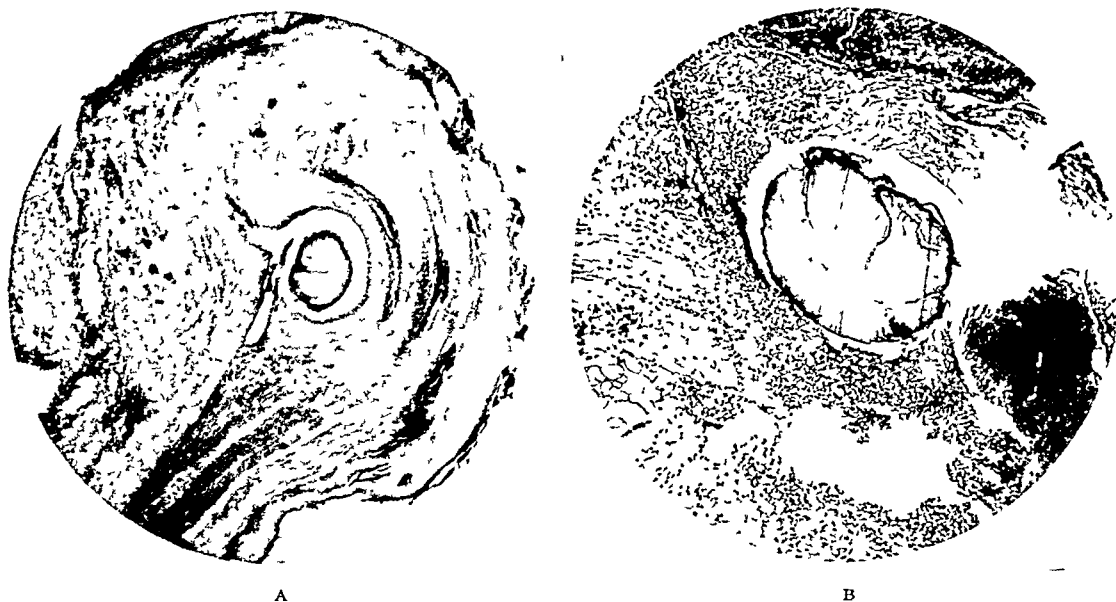
FIG. 7. Forty-eight hours. A, 5-0 chromic—catgut intact; leucocytic infiltration slight. B, o chromic— catgut intact; leucocytic infiltration increased over twenty-four hour section.
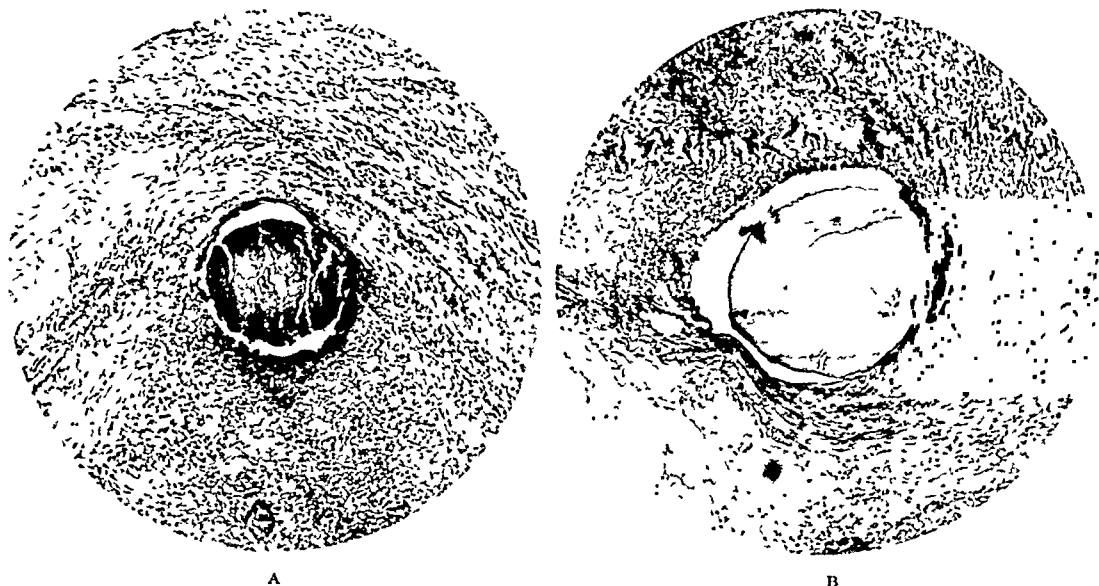
FIG. 8. Seventy-two hours. A, 5-0 chromic—catgut intact; leucocytic infiltration very slight. B, o chromic —cross section of catgut showed beginning disruption; leucocytic infiltration markedly increased.

nique, photographs of specimens, sections of tissue and preservation of specimens were identical with Series II. Specimens were removed at the end of twenty-four, forty-eight, seventy-two, ninety-six and 120 hours.

o *Iodized.* Early necrosis of the mucous membrane, exposure of the catgut strand, persistence of inflammatory reaction involving the mucous membrane approximately 1 cm. distant from the imperfectly united edge, and more marked retraction

of the anastomotic line, were the constant changes accompanying this series.

Summary of Gross Tissue Changes. Min-

were removed and prepared by a modified Spalteholz method, a brief description of which follows:
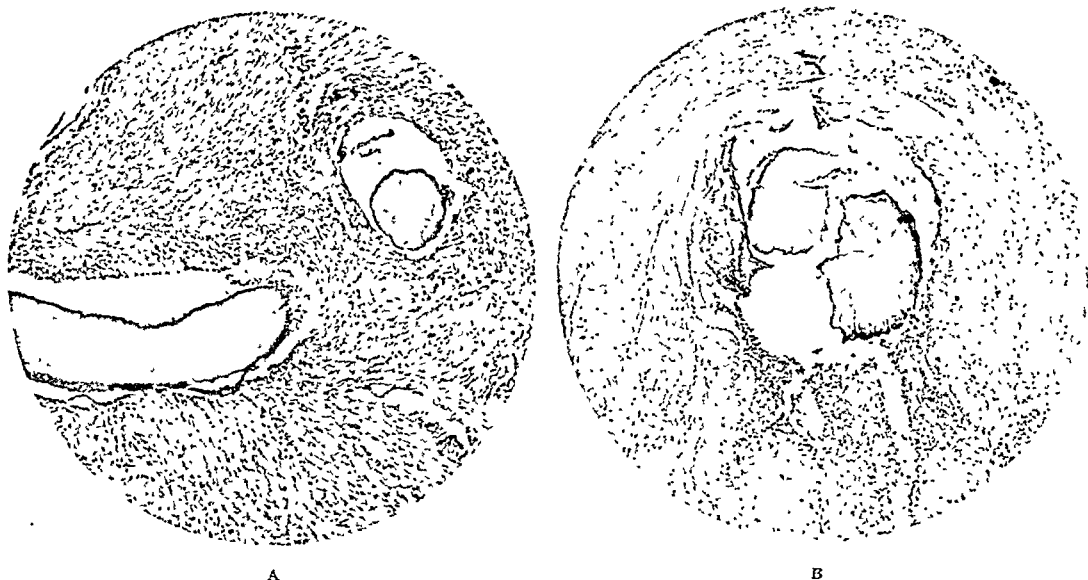
FIG. 9. Ninety-six hours. A, 5-0 chromic—catgut intact; leucocytic infiltration minimal; longitudinal section of catgut showed absence of absorption and minimal tissue infiltration. B, o chromic—cross section of catgut showed marked absorption and increased leucocytic infiltration.

imal mucosal necrosis and inflammatory reaction resulted in immediate, accurate union of the apposed mucosal edges where 5-0 chromic was used. o iodized, on the contrary, showed gross changes similar to and frequently more marked than those observed in the anastomotic line in the stomach where o chromic was used, i.e., early mucosal necrosis, exposure of the catgut strand, inflammatory edema and retracted anastomotic line.

SERIES IV

Five groups of three dogs were prepared as in Series I, II, and III and operated on. Three longitudinal incisions, 7 cm. in length were made in the duodenum between rubber covered clamps, opposite the mesenteric border. Approximately 3 cm. of normal intestine separated the incisions. In each group of three, the serosa, the muscularis, and the mucosa of the first dog were sutured with 5-0 chromic; the second with o chromic; and the third with 5-0 iodized catgut. At twenty-four-hour intervals, as in the previous series, specimens

Chest opened and a cannula ligated into aorta, immediately after death of animals by intrathoracic injections of ether. Vascular channels flushed with saline, then injected with a suspension of barium sulfate colored with ammoniated carmine. Portions of gastrointestinal system containing suture lines removed and fixed in formalin. Trimmed to size, washed thoroughly; bleached by immersion in weak peroxide solution, washed again; gradual but complete dehydration with graded alcohols including two changes by absolute alcohol. Specimen cleared by immersion in chemically pure benzine and pure benzyl benzoate. Preservative or mounting fluid is synthetic oil of wintergreen.

5-0 *chromic* showed minimal evidences of operative trauma, little or no ecchymosis, immediate serosal and mucosal union and maximum outbudding of capillaries throughout the suture series.

o *chromic* showed marked evidence of mucosal trauma in the twenty-four to seventy-two-hour specimens with delayed mucosal union, and a minimal outbudding of capillaries in the ninety-six and 120-hour groups.

5-0 Iodized. This catgut strand was disrupted almost immediately, at least as early as twenty-four hours, in the mucosa tudinal incision in the duodenum with 5-0 chromic, 0 chromic, and 5-0 iodized catgut demonstrate that 5-0 chromic catgut pro-

A   B
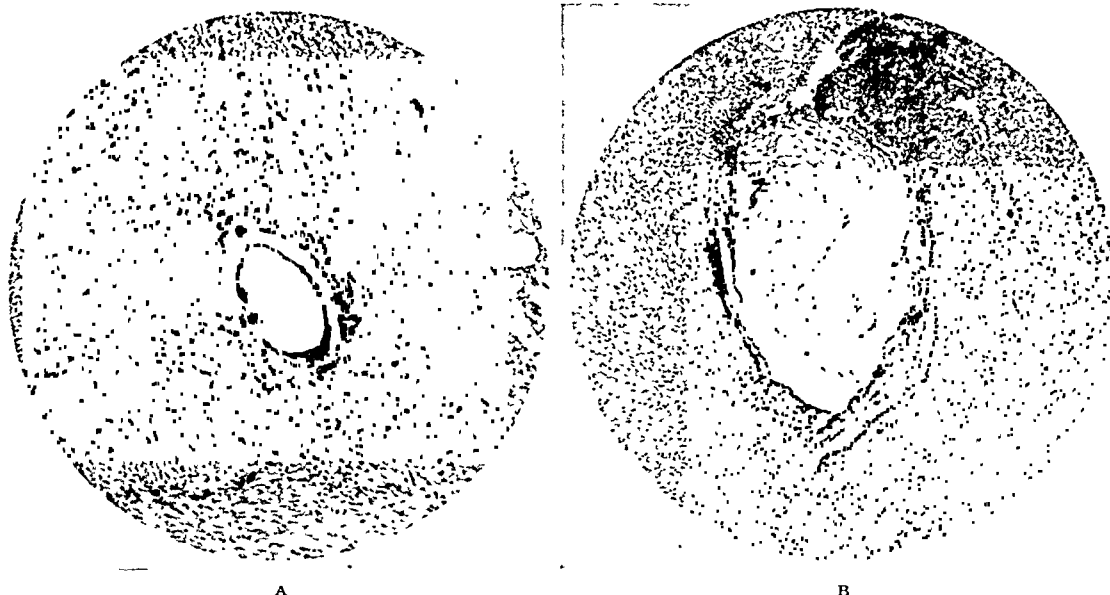
FIG. 10. One hundred and twenty hours. A, 5-0 chromic—catgut intact with minimal tissue reaction. B, 0 chromic—catgut almost completely absorbed; leucocytic infiltration maximal.

A   B
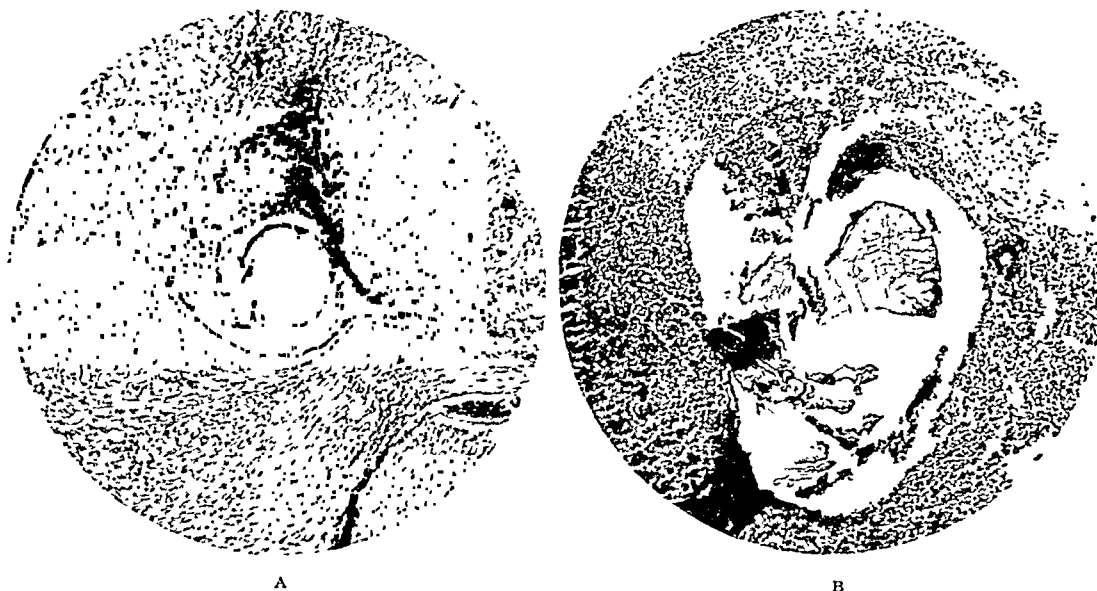
FIG. 11. End of healing period. A, 5-0 chromic—catgut strand intact; slight round cell infiltration. B, 0 chromic—marked disruption of catgut and extensive round cell infiltration.

of the duodenum. All specimens in the series, except the twenty-four-hour, showed complete disruption of the mucosa and partial disruption of the serosa.

Summary of the Gross Tissue Changes. Transparent specimens of suture of a longiduces minimal tissue irritation; that capillary proliferation occurs early resulting in rapid accurate union; that 0 chromic catgut produces marked tissue irritation and slow union of the mucosal surface and that in those specimens where 5-0 iodized catgut

was used, the mucosa in most instances was completely and the serosa partially disrupted.

SERIES V

In five pairs of dogs prepared as in Series I, II, and III, a 5 cm. midhorizontal incision was made in the stomach on the anterior wall, between rubber covered clamps. In each pair the mucosa and seromuscular layer of one dog were sutured with 5-0 chromic continuous catgut; in the other 0 chromic was used. The entire stomach of each dog was removed; one pair at twenty-four, one at forty-eight, seventy-two, ninety-six and 120 hours after the first operation. These specimens were also prepared by the modified Spalteholz method. The stomachs were large and the difficulty of obtaining uniform clearing made it advisable to remove sections of the suture line with contiguous tissue and mount them for comparison.

5-0 Chromic. Transparent specimens of the twenty-four to 120-hour groups inclusive showed that the strand used to unite the serosa remained intact throughout. healing was undelayed due to absence of capillary hemorrhage. The 5-0 chromic catgut strand in the mucosa remained intact for ninety-six hours—the twenty-four and forty-eight-hour specimens showed minimal capillary hemorrhage. The forty-eight-hour specimen, however, showed slight mucosal separation, due to the stretching of the specimen as the catgut strand remained intact with no evidence of ecchymosis. The suture line, however, remained intact. The seventy-two-hour specimen showed beginning capillary extension or budding at the mucosal edges; in the ninety-six-hour specimen the budding had advanced perceptibly. (Plate IIC.)

0 Chromic. Serosal sutures remained intact throughout the series, twenty-four to 120 hours, similar to 5-0 chromic. The mucosal sutures, however, showed absorption taking place more readily than 5-0 chromic, disappearing in the mucous membrane at the end of seventy-two hours.

After ninety-six hours, only the incompletely tied knot of catgut at one end of the anastomotic line remained intact. (Plate IID.) All specimens showed a minimal degree of capillary budding.

Summary of Gross Changes. The transparent specimens present absolute evidence corroborating the conclusions we had reached after microscopic study of the cross sections of catgut; that the finer catgut, 5-0 chromic, remains longer and produces less irritation than the larger, 0 chromic. Capillary budding extends toward and over the anastomotic edge earlier in the 5-0 group, accounting for the solidity of union of the divided mucosa in these groups.

SERIES VI

In the entire study to this point, adult dogs had been used. In order to determine the tissue reaction in young animals and also with the hope of obtaining satisfactory specimens of the entire stomach, we selected six weeks old puppies for the sixth series. Five pairs of puppies were prepared and operated upon, using the same technique as in Series V. A horizontal incision 7 cm. in length was made through the anterior wall of the stomach, and the seromuscular and mucosal layers were sutured with 5-0 chromic and 0 chromic catgut. The modified Spalteholz method was again used in preparing the specimens. The puppies withstood the operative procedure surprisingly well. Specimens were removed at twenty-four, forty-eight, seventy-two, ninety-six and 120 hours after the first operation.

5-0 Chromic. The twenty-four to seventy-two-hour specimens showed minimal or complete absence of extravasation of blood into the mucosal, submucosal and muscular layers of the stomach. In the twenty-four hour specimens a slight brown discoloration on either side of the suture line evidenced slight hemorrhagic extravasation; but in the forty-eight and seventy-two-hour specimens it was completely absorbed and careful inspection

PLATE_II

A

B

C

D
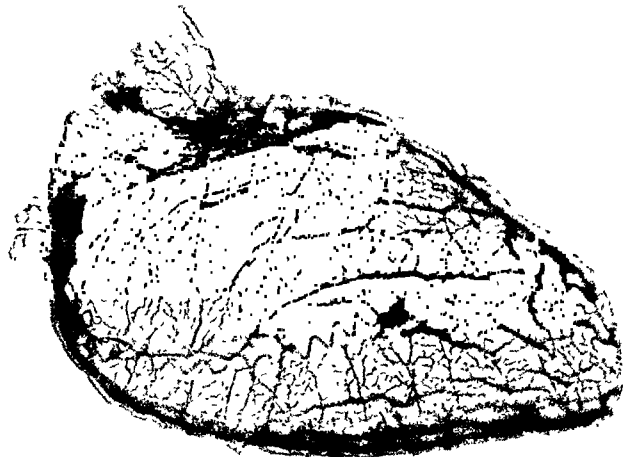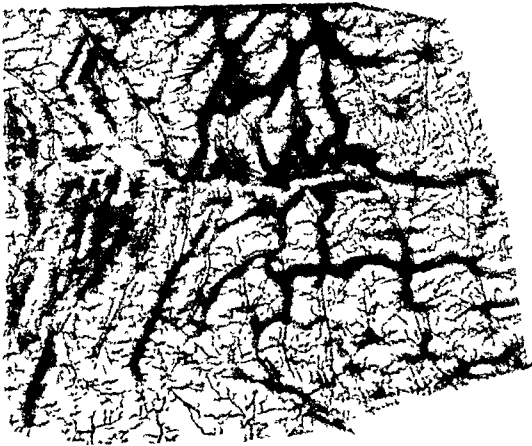
Gross specimens of young and adult dogs' stomachs showing the results of our attempts to obtain better union of the divided mucous membrane, using 5-o chromic catgut. o chromic was used in the control groups.

A, gastric mucosa of young dog united with 5-o chromic catgut, continuous over-and-over suture. Removed ninety-six hours after operation. Because of the very small caliber of the needle and catgut, a minimal number of mucosal cells was destroyed; intracellular extravasation of blood and necrosis with polymorphonuclear infiltration slight. The catgut strand remained intact and the minimal cellular reaction induced is evidenced by the fact that capillaries can be observed approaching, and in some instances actually inosculating with, those across the united mucosa.

B, gastric mucosa of young dog sutured with o chromic catgut, continuous suture. Removed ninety-six hours after operation. Because of the comparatively large caliber of the needle and catgut, excessive destruction of cells occurred; this was accompanied by intracellular extravasation of blood followed by necrosis and polymorphonuclear infiltration. The apposed mucosa was completely absorbed and the catgut strand has almost entirely disappeared—there is no evidence of capillary budding.

The 5-o chromic specimens in the twenty-four, forty-eight and seventy-two hour groups show early absorption of blood and beginning capillary budding at the end of forty-eight hours. In the o chromic specimens the apposed mucosal cells were viable at the end of twenty-four hours, but the excessive amount of blood interfered with the capillary budding and the disruption of the catgut strand began between forty-eight and seventy-two hours.

C, divided gastric mucosa and serosa of adult dog sutured with 5-o chromic catgut. Removed at the end of ninety-six hours. Changes identical with A. Mucosa united; catgut intact.

D, divided gastric mucosa and serosa of adult dog united with continuous sutures of o chromic catgut, removed at the end of ninety-six hours. Mucous membrane did not unite—the edges separated approximately 5 mm.; the catgut was absorbed except the knot at left end of suture line; necrosis of the mucosal edges was complete.

In all four specimens it will be observed that the sutures in the serosa are intact. Because of the slight difference in the points of focus of the mucosal and serosal sutures, the photographer was unable to exclude them.*

* The use of a hand lens in viewing these photographs will aid the reader to see the details more clearly.

with a magnifying glass showed the catgut strand to be unaffected throughout. The ninety-six and 120-hour specimens in this series, along with those of the adult dogs' stomachs, are outstanding among the transparent specimens. They demonstrate the fact (which we were unable to show microscopically) that *5-0 chromic catgut remains in the mucosa longer and produces less irritation than 0 chromic.* Furthermore, capillary proliferation or budding occurs at its maximum rate, the ends of these vessels on either side of the divided mucosa inosculating frequently. This phenomenon was still more noticeable on the fifth day. (Plate IIA.)

o *Chromic.* The twenty-four and forty-eight-hour specimens showed marked evidence of extravasation of blood into the mucosal, submucosal and muscular coats of the stomach; in the seventy-two-hour specimen this was absorbed to some degree, but that it had interfered with the capillary budding was shown by the more pronounced translucent area and at no point did the capillaries cross the line where the apposing edges of mucous membrane were held together. Budding of capillaries, however, had taken place in the 120-hour specimens. (Plate IIB.)

Summary of Gross Changes. Inspection of the transparent specimens of puppies' stomachs with the aid of a simple magnifying glass confirms what was found in the transparent specimens of adult dogs' stomachs. 5-0 chromic remains longer and is less irritating to the tissues of the stomach. Union of the divided mucous membrane occurs in minimum time as shown by the capillary budding. Inspection of the serosa shows that the catgut strand remains unabsorbed when both fine and coarse strands are used; but only the 5-0 chromic remains intact in the mucosa for ninety-six hours—the 0 chromic being disrupted and partially absorbed. (Plate IIA and B.)

SERIES VII

Young dogs were also used in this series to demonstrate the comparative rate of absorption and tissue reaction of 5-0

chromic and 0 chromic in the tissues of the anterior abdominal wall. A vertical 7 cm. incision was made through the middle of the right rectus abdominis muscle; and the peritoneum, muscle and deep fascia sutured. Five pairs of puppies were used, their tissues being sutured, one of each pair with 5-0 chromic and the other with 0 chromic. Sections of the entire suture line including the tissue 2 cm. on either side were removed, prepared by the Kaiserling method, embedded in paraffin, sectioned and stained with hematoxylin-eosin. From four to six sections of each specimen were studied and the ones showing the maximum absorption of catgut in each case was used for photomicrographs which demonstrate a minimal absorption of catgut and tissue reaction in 5-0 chromic as compared with 0 chromic.

5-0 Chromic. Microscopic sections of the entire abdominal wall removed at varying intervals from two to ten days confirm the observations made from the microscopic sections of the stomach. 5-0 chromic catgut remained intact and produced a minimum of tissue irritation. At the end of ten days, the strand was still intact and the tissue around it showed minimal leucocytic infiltration.

o *chromic,* as in the gastric specimens, began to disintegrate on the third day and increased until the catgut was practically absorbed on the sixth to tenth days. Leucocytic infiltration increased commensurately.

Summary of Microscopic Changes. 5-0 chromic and 0 chromic catgut were used to suture the peritoneum, rectus abdominis muscle and deep fascia of young dogs. Cross sections of tissue removed and studied microscopically at periods varying from two to ten days revealed that the 5-0 chromic strand remained intact longer and produced minimal tissue irritation as compared with the larger 0 chromic catgut strand.

RESULTS

Comparative study of microscopic changes occurring in the muscle, fascia,

and peritoneum of the abdominal wall, and gross changes in the mucous, submucous, muscular, and peritoneal coats of stomachs of young dogs shows that 5-0 chromic catgut produces minimal tissue reaction and maintains integrity longer than o chromic catgut.

In the stomach, absorption of o chromic catgut begins forty-eight hours after operation; is almost complete in five days, and is attended with maximal neutrophilic mobilization. Integrity of 5-0 chromic catgut is maintained throughout the healing period, and leucocytic infiltration is minimal.

Comparative study of gross tissue changes in the suture lines accompanying gastroduodenostomy, incision and suture of the ileum of young dogs, and incision and suture of the stomach of mature dogs, shows in every instance the superiority of 5-0 chromic catgut. It produces minimal tissue reaction and maintains integrity longer than o chromic catgut. The 5-0 iodized catgut and the o iodized show early disintegration.

CLINICAL INVESTIGATIONS

The results of our experimental studies were verified by clinical investigations. The practical value of this new fine-size suture of 5-0 chromic catgut was repeatedly demonstrated in surgery of the gastro-intestinal system and biliary tract; in suturing the dura, tendons (tenorrhaphies), and nerves (neurorrhaphies); and in the surgery of children. Complete report of clinical results will appear later.

CONCLUSIONS

Our experimental investigations and clinical observations have demonstrated that 5-0 chromic catgut may be used to replace larger sizes of sutures in a variety of surgical situations. Where close and uniform apposition, together with minimal trauma and cellular reaction are essential, it is especially indicated.

The use of 5-0 chromic catgut markedly reduces tissue trauma; produces minimal cellular reaction; insures strong apposition of mucous and serous membranes, because nearly twice as many stitches can be inserted within a given space, thereby providing a suture line of great strength; gradual and uniform absorption insures prolonged retention of the catgut in the tissues, and thereby reduces danger of hemorrhage and peritonitis.

SIMPLIFICATION OF THE PHYSICK-FRANK-SELLHEIM PRINCIPLE OF EXTRAPERITONEAL CESAREAN SECTION*

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IN Dewees[1] text on midwifery there appeared a letter (as a footnote) dated September 28, 1824 and written by William Edmonds Horner, then Adjunct Professor of Anatomy at the University of Pennsylvania. This letter credited Philip Syng Physick, Professor of Surgery at that institution, with a new principle for a true extraperitoneal cesarean. Horner stated in part that "the peritoneum be stripped from the upper fundus of the bladder by dissecting through the connecting cellular substance." Physick even advised moderate distention of the bladder to facilitate visualization of that viscus and to promote ease of dissection.

This reference passed unnoticed. Indeed, the few efforts at extraperitonealization during the remainder of the nineteenth century—those of Baudelocque,[2] Cianflone,[3] Thomas,[4] Skene,[5] Budin,[6] Hime,[7] Edis,[8] Gillette,[9] Dandridge,[10] Jewett,[11] Poullet,[12] were all of the Ritgen-Baudelocque-Bell type. Singularly enough, of these fourteen operations, six were performed in Brooklyn, New York. Ritgen[13] attempted the first extraperitoneal cesarean (October 12, 1820) by incising parallel to the right Poupart's ligament, from midsymphysis to the crest of the ilium. The "loosely fitting" peritoneum was displaced and the vagino-cervical (lower segment) area incised. This technique, an innovation, proved a failure in Ritgen's hands; the severity of the hemorrhage and the patient's collapse interfered with its completion. The procedure was labelled *gastro-elytrotomy* or *laparo-elytrotomy* (Bauch-scheidenschnitt) by Ritgen. Baudelocque wrote his Paris thesis on this subject in 1823 and Charles Bell,[14] Professor of Surgery of Edinburgh, outlined the steps of the operation in his

Institutes of Surgery (1838). Adequate reviews of this type of cesarean were subsequently written by Garrigues,[15] Masson,[16] McCormack[17] and Clarke.[18]

The first to attempt an extraperitoneal cesarean according to the suggestion credited to Physick was Frank[19] of Cologne in 1907. He pointed out that if the "bladder was high" it was possible to strip the peritoneum from the vault of that viscus. Frank, though successful in the occasional case, met with considerable difficulties in separating the peritoneal fold from the bladder dome. These same difficulties were encountered by Sellheim[20] in the following year (1908) in his numerous efforts to find a true extraperitoneal approach. The principle was completely abandoned when Latzko[21] introduced his method of extraperitonealization by lateral displacement of *both* the bladder and the peritoneal reflection (1909).

The difficulties encountered by Frank and Sellheim were due, primarily, to their persistent attempts at beginning the separation of the peritoneal fold from the bladder wall exactly in midline, which is the maximum point of fusion (urachus) between these two structures. These difficulties, the author has circumvented in a simple way—by combining the Frank-Sellheim method with the Latzko lateral approach. This article is presented merely to outline the simplification of an old principle, thereby rendering that principle surgically applicable and useful. The procedure is not a distinct innovation, but rather a combination of steps (all previously utilized) which facilitates a true extraperitoneal approach to the lower segment, thereby inflicting a minimal amount of surgical injury on the patient and avoiding

the hazards of surgical trauma to the bladder. The operation is applicable both to the laboring and non-laboring patient,

With the completion of these steps, the patient is placed in a moderate Trendelenburg position. A Pfannenstiel incision,

I

Franks »Suprasymphysäre Entbindung«

II

Sellheims »Extraperitonealer Uterusschnitt«
(Sellheim I)
Abgelöster Peritonealuberzug des Blasenscheitels und Plca
nach oben verschoben

III

Meine Methode
Blase nach rechts verlagert.

IV

Meine Methode
Blase seitlich abgelöst und verzogen Ligam ves co umbil lat.
durchschnitten
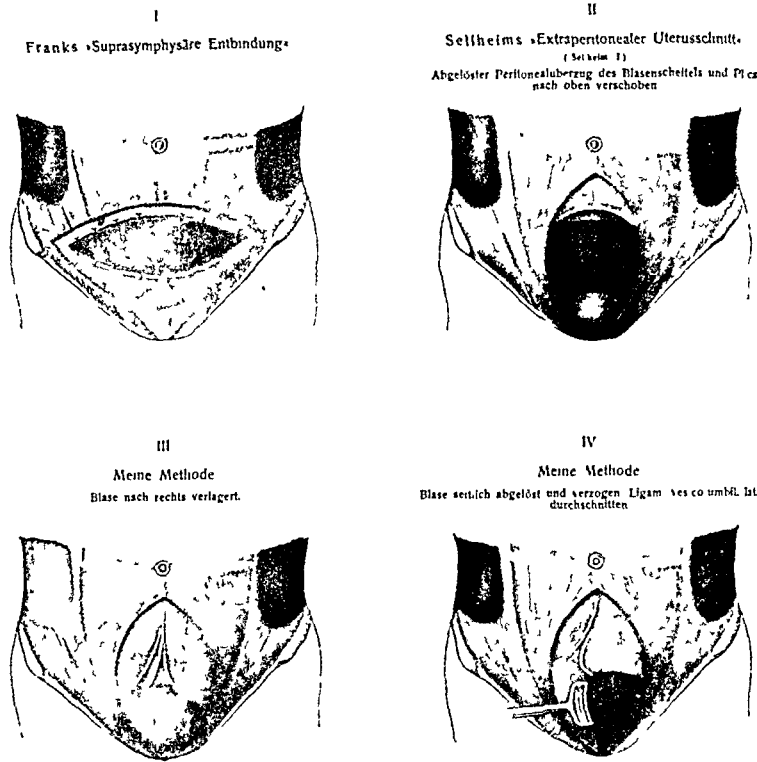
FIG. 1. I, Fritz Frank's peritoneal exclusion operation (1907). II, Hugo Sellheim's extraperitoneal cesarean (1908). III and IV, Wilhelm Latzko's extraperitoneal method (1909). (From *Wien. klin. Wcbnscbr.*, 1909.)

although, in the latter instance, the dissection must be conducted with greater surgical delicacy and technical skill.

Technical and Anatomic Considerations. The patient is placed on the operating table with extremities in lithotomy position. After adequate vulval aseptic preparation, the bladder is emptied with a number 16 F. catheter which is inserted for two-thirds its length and left in situ. This measure will delineate the contour of that viscus in a more practical manner than vesicoclysis. The vagina is packed, rather thoroughly, with iodoform gauze, for the purpose of displacing the inferior portion of the lower segment (and the fetal head if needs be) upwards above the symphysis, thus bringing the essential parts squarely into the operating field.

$4\frac{1}{2}$ inches long, is made just above the pubic hairline, preferably in the normal skin fold usually found in the suprasymphysial area. With proper retraction and lateral deviation of the recti muscles, the parietal *fascia transversalis* appears in the operating field. *This structure is incised transversely, well over the bladder surface, avoiding that part of the fascia covering the parietal peritoneum; since it (the fascia) serves as a reinforcement of that otherwise exceedingly thin peritoneal membrane.* This is the "key" to the success of the operation. Incision of the fascia covering the parietal peritoneum invariably leads to a break in the contiguity of that membrane. It was this initial error which (in part) accounted for the dissectional difficulties

and the failures encountered by Frank and Sellheim.

It is well to recall that the pregnant

is carried upward, i.e., *cephalad*, by the gravid uterus and it becomes an abdominal rather than a pelvic organ, even in its non-
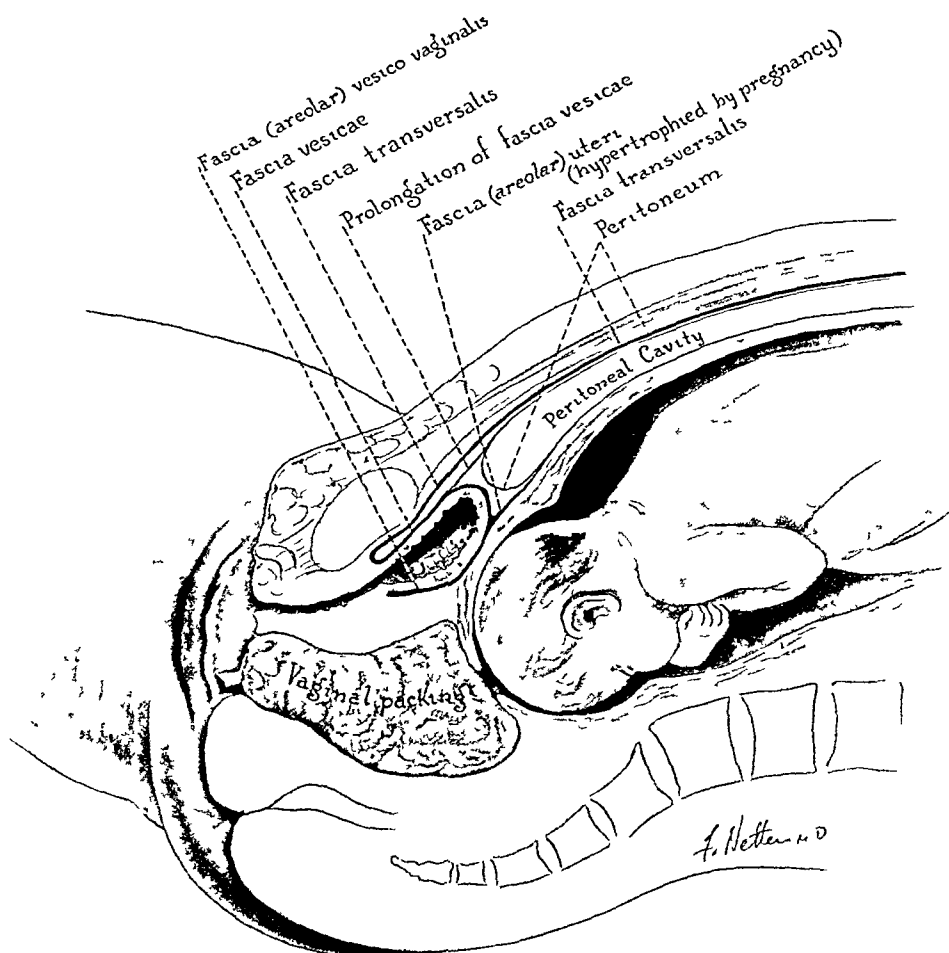
FIG. 2. Semidiagrammatic demonstration of the arrangement of the fascia in relationship to bladder, lower uterine segment, parietal peritoneum and peritoneal fold

woman *at term* presents certain distinctive changes in texture, shape, size and relationship involving the bladder, uterus, peritoneum and fascia. These structures may participate in all of these changes to a variable degree or in only one of them to a total exclusion of the others. And it is these changes in anatomic relationships which render this operation feasible. First, the peritoneum—including that part juxtaposed and adherent to the uterus—though it does not manifest the usual hypertrophies and engorgements of the genitalia, changes its shape and relationship with respect to the bladder and the uterus. Second, the bladder

distended state. Third, the parietal *fascia transversalis*, a thin yet dense inelastic membrane, beneath the symphysis, begins its devious extensions to, and reflections over, the pelvic organs and the bladder. That portion of the fascia on the anterior vesicular wall (*fascia vesicae*) loses its fibrous quality and becomes a thin frail covering of a nonsupportive nature. This fascia is *not* adherent to the bladder; the intervening space is padded with a mesh of engorged fatty pellicles—fat which adheres to the bladder. As the *fascia vesicae* approaches the posterior surface of the bladder and the anterior aspect of the

lower segment, it again changes and becomes a loosely knitted fibro-areolar layer markedly hypertrophied, particularly

mental area during the strenuous stages of labor.

It is important to recall that the indis-
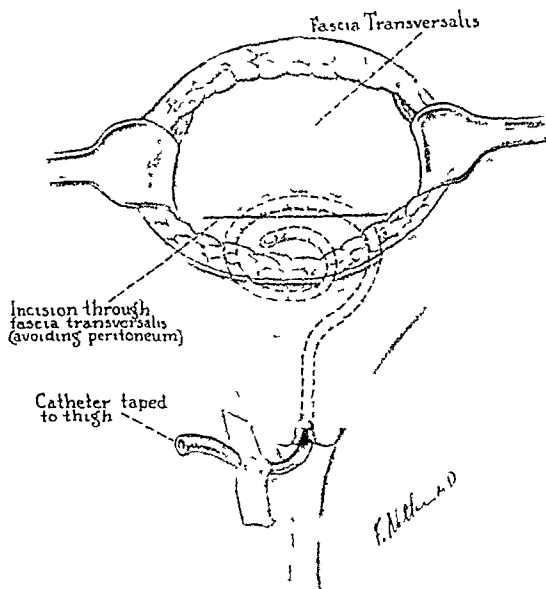
FIG. 3. Pfannenstiel incision, lateral deviation of recti, fascia transversalis, bladder with catheter in situ and incision of fascia transversalis over the upper surface of the bladder.
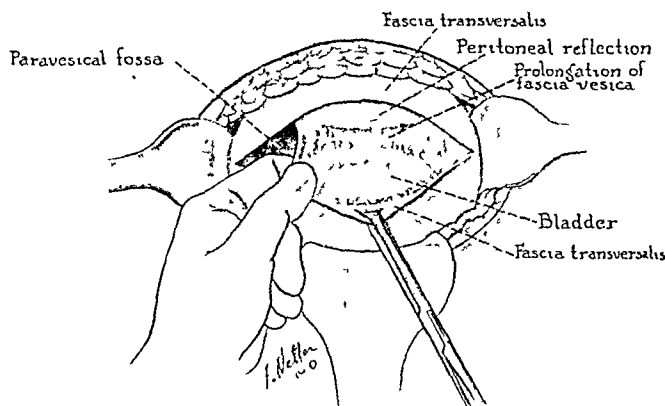
FIG. 4. Incised fascia transversalis, exposed peritoneal fold, dome of the bladder with catheter, prolongation of fascia vesicae and finger in the paravesical fossa.

in the terminal stages of the gravid state. This hypertrophy must be considered a buttress of appreciable value to the attenuated musculature of the lower seg-

criminate use of the term "fascia" and the controversial data on its texture and distributions in the pelvis have led to a maze of misunderstanding, and have left

the present day gynecologists of the various schools of vaginal plastic surgery without a common anatomic denominator. This by Jacob Henle, the great German histologist and anatomist. A description and hand drawing of the microscopy of the
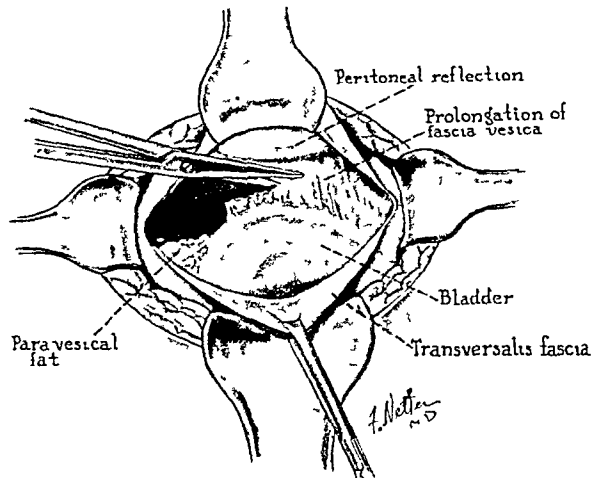
FIG. 5 Bladder elevated from its bed, incision of connecting fascia between peritoneal fold and bladder dome.

medley of tangential views has been the result of the many contributions based on gross anatomic dissections without any histologic corroboration. The true relationship of the *fascia endopelvina* to the pelvic organs and its variations from a fibrous

bladder wall and vaginal wall with the intervening areolar strands appeared in his *Handbuch der Systematischen Anatomie des Menschen* (1866–1871). This drawing was reprinted in the first "system" of American gynecology published in 1887 in the chap-
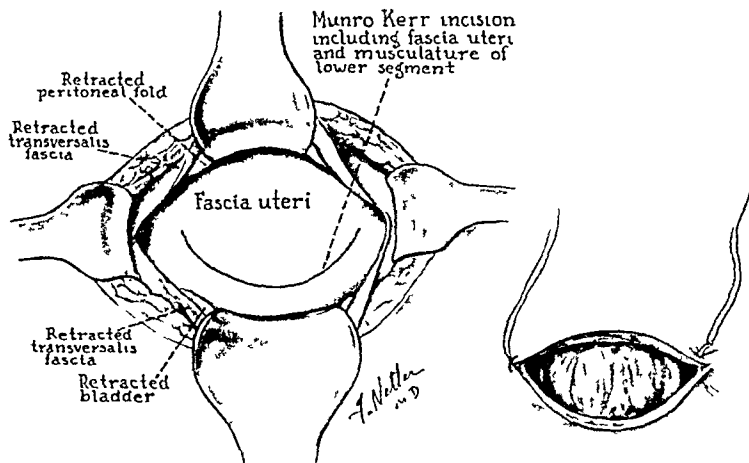
FIG. 6. Exposed lower segment and Munro Kerr incision. (Insert—ties in apices of incision and presenting fetal head.)

sheath to a loose areolar pad was painstakingly studied and described (histologically) by B. H. Goff[22] (1931). The existence of this avascular line of cleavage with strands of areolar tissue was first mentioned

ter on pelvic organs by H. C. Coe. It was lost sight of until redescribed by W. Blair Bell in his text on gynecology (1912). P. C. Huguier had, however, demonstrated the existence of this space between vagina and

bladder in a drawing (macroscopic) which appeared in his text, *Sur les allongements hypertrophique du col de l'uterus*, 1860.

above the internal os, and both lose their identity.

In the normal (non-pregnant) state the
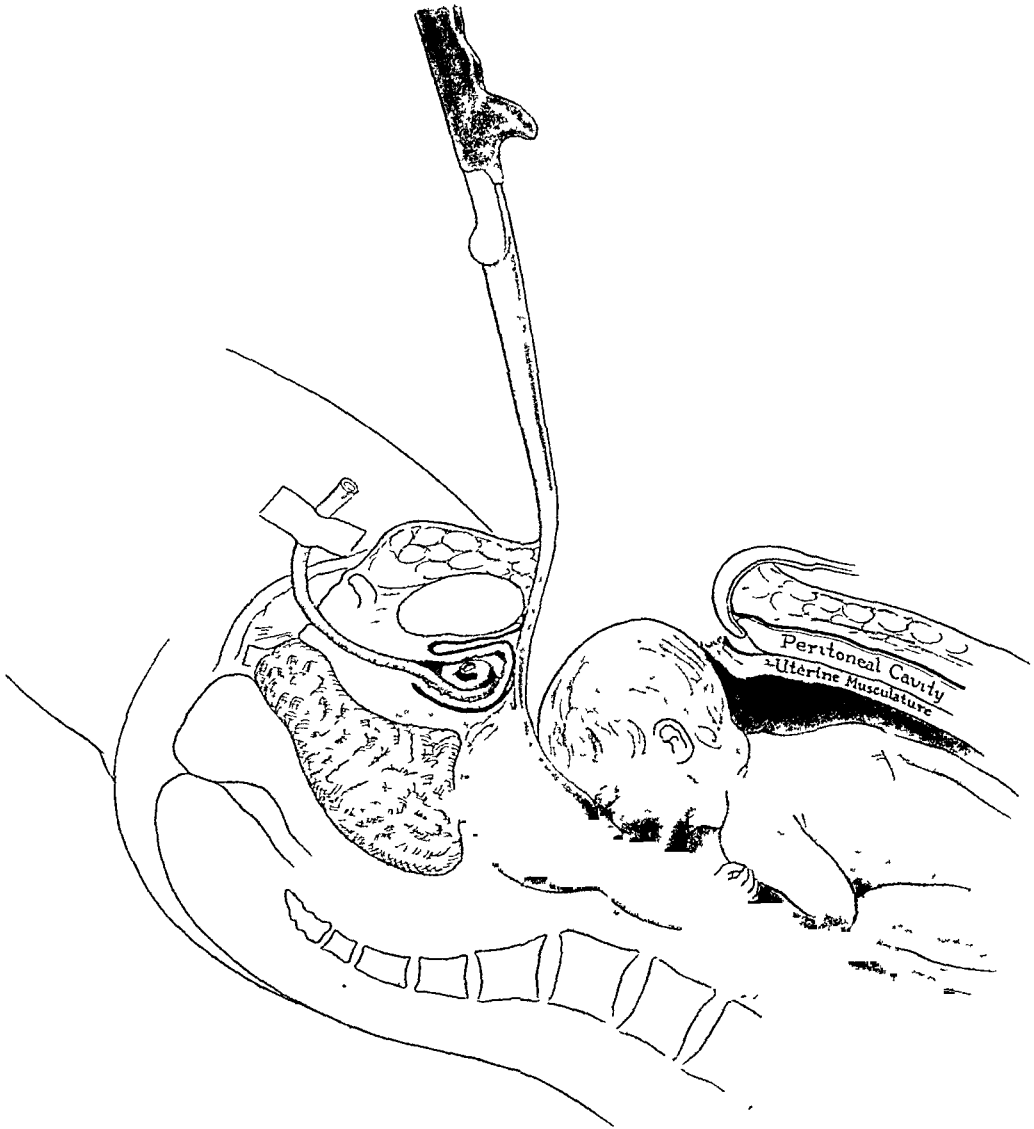
FIG. 7. Delivery of head by use of blade as a vectis.

That part of the *fascia transversalis* which descends into the pelvis has been designated as *fascia endopelvina*—with even more detailed and appropriate designations as *fascia vesicae, fascia uteri, fascia vesicovaginalis* and *fascia rectovaginalis*. The *fascia uteri*, like the visceral peritoneum becomes fused with the uterine substance approximately in the area just

posterior surface of the bladder with its peritoneal covering help to form the vesicouterine excavation. This excavation is gradually eliminated in the terminal months of gravidity, as the flattened bladder ascends the lower segment, and the peritoneal fold recedes into an elongated cul-de sac. This recession permits the formation of an avascular space bounded

by the apex of the bladder below, by the peritoneal fold above, by the prolongation of the *fascia vesicae* anteriorly and by the lower segment posteriorly. This space must be entered in order to bring the lower uterine segment extra-abdominally.

Re-reading this anatomic description with the diagram as a guide, reveals the all important fact that the bladder lies free in a cleavage space, encased in the *fascia vesicae* (save at the urethral area), below the parietal *fascia transversalis* and above the hypertrophied layer of the loosely knitted uterine portion of the *fascia endopelvina*, padded anteriorly and (particularly) on either side with a loose mesh of fatty tissue. Entering this space at the right lateral border of the bladder (the paravesical fossa), this viscus is separated by blunt dissection with index and middle fingers—separated from the sublying *fascia uteri*. With this separation completed, the bladder is gently elevated from its bed, resting on the palmar surface of the two fingers of the left hand. This maneuver brings into clear relief the bladder dome outlined by the catheter, the grayish white peritoneal reflection and a thin intervening transparent tissue—the hypertrophied prolongation of the *fascia vesicae*. This tissue is "scissored." At midpoint, the transparency fades as the peritoneum is reflected in a series of folds on the middle umbilical ligament (urachus) and on the obliterated hypogastric arteries. Nonetheless, a definite line of demarcation is visible; because in late pregnancy these folds and cords or false ligaments of the bladder are flattened and attenuated. Cognizance of this embryologic-anatomic relationship, coupled with an added dissectional care, will aid in avoiding peritoneal or bladder trauma. Thus a complete separation of the bladder dome from the peritoneum can be accomplished effectively without injury to either structure, and the lower uterine segment, with its fascial covering, is exposed extraperitoneally. The exposure is further improved by displacing the bladder down-

ward behind the symphysis with a Doyen retractor.

This segment is incised in semilunar fashion (Munro Kerr incision), beginning in midline and as low as possible in order that the extremities of the incision may remain in the lower segmental area. Bleeding points arising from the cut ends of the musculature may be controlled by the use of Allis or Péan triangular clamps. The two ends of the incision are sutured and the sutures are held long to facilitate subsequent exposure of the cut edges and repair. The fetal head is rotated manually to a direct vertex anterior position. A solid blade (preferably the left blade of a Tucker-McLean forceps) is used as a vectis, resting on the midpoint of the symphysis acting as fulcrum. By this maneuver, and with added pressure exerted on the flanks of the patient, the fetal head is eased out and delivered. At this point it may be found advisable to delay cutting the cord for a moment while the anesthetist permits the patient to breathe an adequate amount of CO_2. This will stimulate the anesthetized infant's respiratory apparatus.

The placenta may be delivered either by a manual invasion of the fundus, if the operator is dealing with a "clean" case; or, it may be removed by traction on the cord and compression on the fundus extra-abdominally, when dealing with a potentially infected case. The author has found it advantageous to pack the uterus in the majority of instances with a moderate amount of 2 inch iodoform gauze. Assurance is made that one end of the gauze is placed at the cervical orifice to facilitate removal. Occasionally, in the cesareanized patient with an undilated cervix, this packing will soften that structure, dilate it and permit freer (sapremic) drainage. This gauze is removed in approximately thirty-six postoperative hours. The subcutaneous injection of five minums of pituitrin precedes the removal. The uterine wound is sutured in two layers with continuous chromic No. 2 catgut—the first involving the musculature; the second, the muscula-

ture and *fascia (uteri) endopelvina*. The bladder is replaced in its bed and the cut edges of the fascia transversely approximated with interrupted sutures. No drainage is necessary.

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WE are not all alike in the presence of pain. Popular opinion in this respect is by no means at fault. . . . But, medicine has assumed that these differences in respect of response to pain are due to differing grades of will power and of character; when they depend most certainly on many other things yet to be determined.

From—"Surgery of Pain" by René Leriche (Williams & Wilkins).

A MODIFICATION IN THE REPAIR OF COMPLETE LACERATION OF THE PELVIC FLOOR

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AT times perfect operative technique in the repair of a third degree laceration of the pelvic floor is vitiated by a spontaneous breaking down of the repaired rectal wall. Also accidents do happen, and an enema (despite orders to the contrary) may be given after operation, with very untoward results.

For this reason I modified the repair of rectal tears several years ago so that the above described complications cannot arise. This operation has given me very satisfactory results.

Figure 1 shows the posterior vaginal wall pulled forward, the levators exposed, and the rectum with its tear and torn sphincters evident. Now, instead of suturing the tear, the rectum is further mobilized (which is nearly always easy) until the upper end of the tear can be pulled down freely to the perineal surface. The flaps so produced on either side are excised as indicated, and three 40-day chromic No. 2 catgut sutures are placed along the border of the anterior rectal wall and left long, to facilitate pulling the rectal wall into proper position after the torn sphincter ends have been approximated. The levator muscles and the torn ends of the sphincter are then united in the usual way with interrupted chromic No. 2 catgut sutures. The long sutures on the rectal wall are threaded and
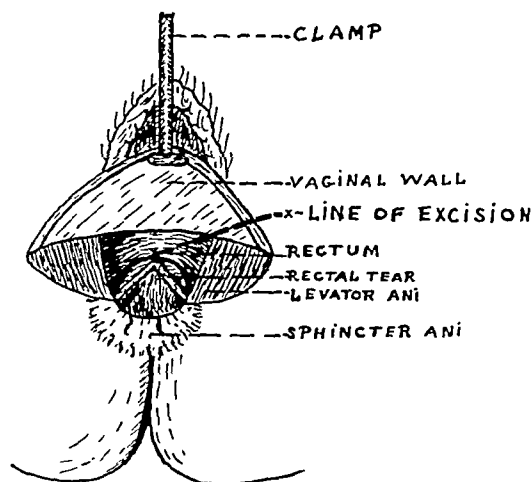
FIG. 1. Repair of complete tear of pelvic floor.

sutured to the new anterior mucocutaneous junction of the sphincter. The perineorrhaphy is then completed in the usual way.

The advantages of the described procedure are that there is no sutured gut buried deeply below the surface, and even should an enema be given, as was done in one case, the consequent ballooning of the gut does not pull on the suture line, and consequently will probably do no damage.

THE USE OF FASCIA LATA IN VAGINAL SURGERY

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MANY operations have been devised for rectocele, and all of these give satisfactory results when the recto-

FIG 1. High rectocele bulging forward.

cele or rectovaginal hernia is low in the vagina. The operation herewith described is particularly designed for high rectocele. It may also be used to repair enterocele, with or without obliteration of the sac.

The operation is done under gas-ether anesthesia. Fifty c.c. of blood are removed from the patient, and sodium citrate is added. This is then placed in a warmed, sterile dish, which is to hold the fascia strip. Warm saline may be used instead, if preferred.

One strip of fascia is removed from below upward, with the modified Masson fascia stripper, as in hernia operations. The strip is split longitudinally and the segments placed in the basin holding the patient's blood (or in warm saline).

The vagina is prepared and a transverse incision made in the perineum, as in typical perineorrhaphy procedures. The vaginal
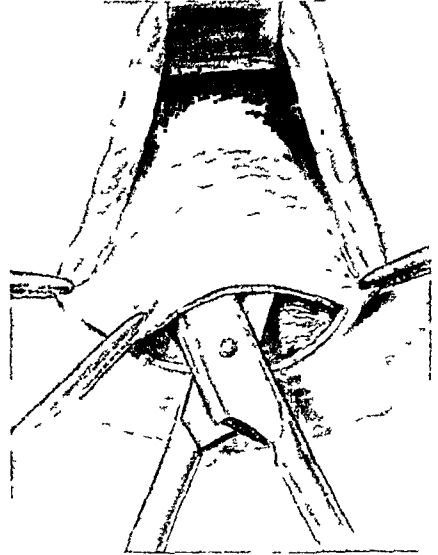
FIG. 2. Preparation of vaginal mucosa, and separation from rectal wall as high and wide as possible.
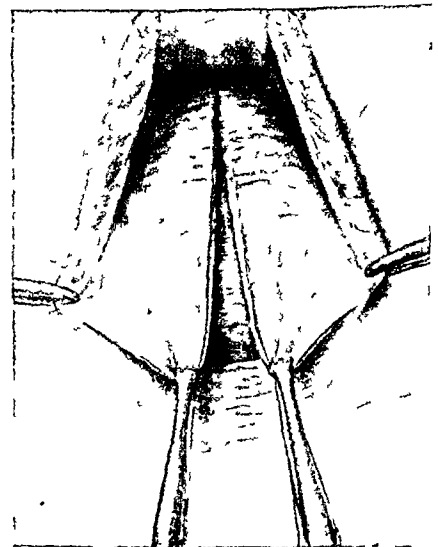
FIG. 3 Longitudinal incision in mucosa to the cervix or vault of vagina, and complete freeing of flaps.

mucosa is freed with scissors and by blunt dissection to the top of the posterior vaginal fornix. A longitudinal incision is

plication markedly improves the condition. Two fascia strips are then inserted in figure-of-eight sutures, using Galle fascia
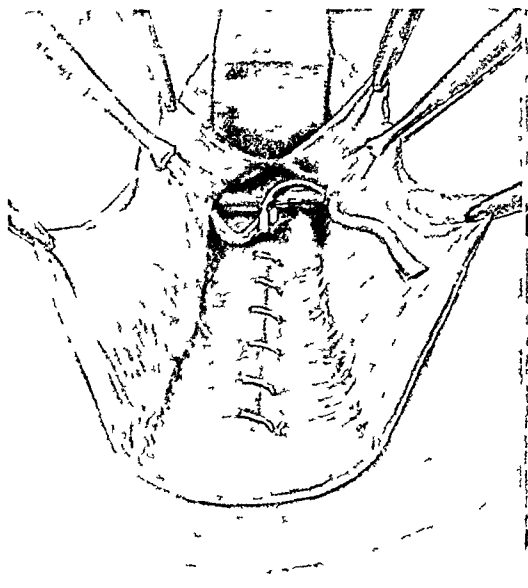
FIG. 4. Fascia lata strip being sutured in place after plication of rectal wall. (Fascia kept in warm saline or citrated blood.)
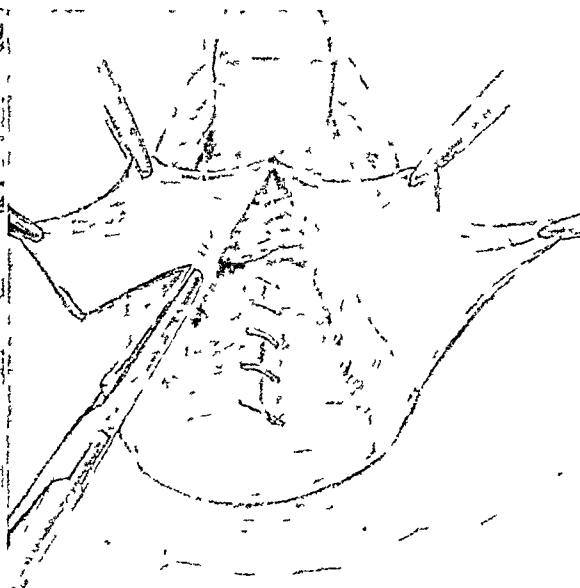
FIG. 5. Two or three fascia lata strips may be used to give adequate support to upper part of rectocele.
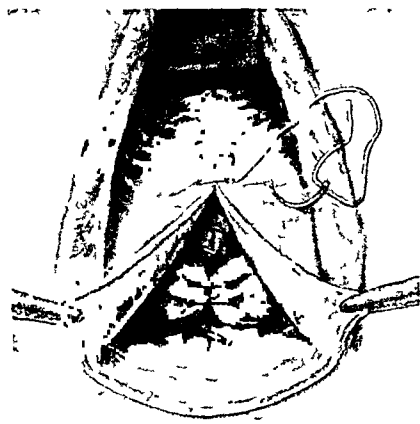
FIG. 6. Vaginal mucosa sutured. Levators approximated in midline.

FIG. 7. Completed operation. Catgut used throughout.

made the entire length of the vaginal mucosa to its attachment in the vault. With blunt and sharp dissection, the mucosa is freed to the lateral walls. The entire rectum now bulges into the wound.

Beginning at the top, the wall is plicated with fine continuous catgut (chromic No. 0) on a curved intestinal needle. This

needles to the lateral pelvic fascia. The fascia sutures are tied and the knots sutured with No. 2 chromic catgut.

The excess vaginal mucosa of the flaps is removed. The upper two-thirds of the

incision in the mucosa are sutured with interrupted No. 2 chromic. The levators with fascial coverings are exposed and sutured in midline. A few interrupted sutures close the perineal fascia and skin.

A self-retaining catheter is inserted and allowed to remain for four days. Two tablespoons of mineral oil are given daily and a fluid and bland diet is maintained for the first five days after operation. At the end of this period, a 4 ounce olive oil enema is given.

A firm posterior vaginal wall throughout is obtained as a result of this operation. Rectal examination shows a solid anterior wall.

The fascial procedure may also be used in the repair of cystocele, fistula, and urinary incontinence. Possibly ox fascia may prove as suitable as that of the patient.

THE special susceptibility to pain of certain people, their natural hyperesthesia, even their pains, are to be regarded in terms of their individual sympathetic equivalents, depending, indeed, upon a habitual heightened vaso-motor tone, in virtue of which their nerves are continually on the threshold of suffering and of pathology.

From—"Surgery of Pain" by René Leriche (Williams & Wilkins).

THE ENDOCRINES AND SPERMATOGENESIS*

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THE rapid development of new information concerning the endocrines has resulted in the accumulation of a large and rather confusing literature. The relationship between the internal glands and the male genital system is of particular importance to the urologist because of the growing appreciation of its application to male fertility.

It is conceded that the pituitary is involved in all matters of endocrine function, and hence, it has been rightly designated as "the master gland." The basophilic cells of the anterior lobe are thought to have particular domain over gonadal development and activity, and from this lobe arise two hormones which have definite gonadotropic qualities.[1] By this it is meant that these substances have the ability to stimulate the gonads of either sex. One is called the follicle stimulating hormone or prolan A, which stimulates follicular development in the ovary, while the second is known as the luteinizing hormone or prolan B, which luteinizes the ruptured follicle. In the male they act on both the interstitial and spermatogenic cells.[2] Both of these agents may be extracted from the anterior lobe of the pituitary by alkaline solutions, but most observers believe they have not yet been isolated as separate and distinct entities.

In the normal human, gonadotropic hormones are contained in the blood and urine of the pregnant woman following production in the chorionic tissue of the placenta. A pregnancy urine extract is capable of producing an effect which is practically identical with the prolan B fraction of the anterior pituitary gland. Experiments indicate that it probably never stimulates follicular development in the female, and in the male causes only functional activation and hypertrophy of the interstitial cells of the testes. This is particularly apparent in studies on prepubetal animals.

Likewise, gonadotropic hormones are obtained from the blood and urine of the castrate male or female, most commonly, from menopausal urine. This has predominantly follicle stimulating power, and also activates the spermatogenic cells of the seminiferous tubules of the male.[3]

To summarize: There are three principal types of gonadotropic hormones in the normal human: those from the pituitary, the urine of pregnancy, and the urine of castrates. All contain varying amounts of the two fractions, but differ in their mode, degree, and type of action. Their chemical behavior suggests protein-like characteristics, but none has been completely isolated or synthesized.

A fourth source of gonadotropic substance in the human is associated with certain neoplasms of the testicle. Aside from the great clinical value of the now famous Aschheim-Zondek test for the detection of these tumors, this phenomenon has little applicability to the topic under discussion. Mention should also be made of extract of pregnant mare's serum, because of its similarity to castrate urine in hastening development of the follicle.

The identification and recognition of the characteristics of these substances has largely been accomplished by animal studies upon the rat and monkey. It is conceded that different species exhibit endocrine reactions which are not paralleled by the human. The action of the gonadotropic hormones on the ovaries of animal

and man has received greatest attention, but Smith and Engle have contributed important conclusions from studies of the male rat. If the pituitary gland is removed, involution of the germinal cells of the testes tubules results until there is a complete regression of spermatogenesis at the end of twenty-one days. When the hypophysectomized animals are treated with pregnancy urine extract there is a partial restitution of germinal tissue. When treatment is combined with castrate urine extract a remarkable regeneration follows.

Additional evidence of the close relationship of spermatogenesis and the gonadotropic hormones has been advanced by Wells and others in the study of rodents.[4] The male squirrel has seasonal breeding habits, and during estrus has increased amounts of gonadotropic material in the blood and urine. Spermatogenesis can be induced out of season by supplying these hormones.

There is, therefore, evidence at hand which indicates that appropriate use of gonadotropic substances can cause hyperplasia and hypertrophy of testicular cells in hypophysectomized animals and that they appear to be closely related to the onset of spermatogenesis in seasonal breeders.

The testicles themselves manufacture internal secretions which are known as androgens.[5] Research has been fortunate in isolating pure endocrine compounds from the gonads, some of which have now been synthesized from the cholesterol derivatives. It is possible, therefore, to check the presumed function of the gland with pure uncontaminated principles. The first androgen recovered from male urine was androsterone. Later, testosterone was isolated in pure form directly from the testicle, and it can now be prepared synthetically. Ružička states that testosterone is twenty-five times more powerful than androsterone. Both substances stimulate comb enlargement in the capon and have direct relation to the growth and functional activity of the prostate and seminal vesicles.

It is noteworthy that both sexes contain male and female sex hormones in their blood and urine. Women normally excrete two-thirds as much androgen (androsterone and dihydro-androsterone) as do men; whereas, the normal male has about one-half the amount of female sex hormone in the urine as do women.[6]

In male castrates, the amount of excreted androgens is reduced to one-third normal. Androsterone and testosterone act differently in certain species, but it is now quite clear that in the monkey and human they have no ability to support or stimulate spermatogenesis.[7]

The thyroid is linked into the endocrine chain and a relationship of some kind exists between it and the organs of reproduction. This gland becomes enlarged in women at puberty, during menstruation, and with pregnancy. Myxedema is accompanied by sexual depression of both sexes. Little precise information is at hand, however, to explain its exact rôle, and it is probable that its influence on the testes is an indirect one.

Adrenalectomy in the male rat causes a loss of libido and potency, as well as degeneration of the seminiferous tubules. There is no convincing evidence that excess of the adrenal principle overstimulates gonad function.

Finally, it is necessary to call attention to Collip's theory of the anti-hormones. These agents are thought to stabilize or immunize the endocrine system, so that subsequent injections of hormones become decreasingly effective. It has become apparent that there is probably a "species specificity." However, this entire mechanism may be a foreign protein immunity. In other words, man exhibits no anti-hormone production against endocrine products derived from man.[8]

The health of the individual is directly dependent upon the proper functioning of the endocrine system. Disturbances which molest the usual balance of the hormones may cause profound anatomic distortions, pathognomonic of a particular type of en-

docrine dyscrasiä. Gigantism, hypogonad-ism, and Fröhlich's syndrome as familiar examples of such dysfunction. Sterility is often an essential characteristic of some of these types, for the gonads readily share the effects of hormonal disturbances.

Lesser degrees of endocrine disorders may produce minor changes, causing in-conspicuous alterations which may remain unappreciated until there is occasion to investigate a particular organ. It is now known that certain men are sterile, yet in other respects they have all the requisites for complete masculinity. They have well developed secondary sexual characteristics, faultless libido, normal sexual ability, and no appreciable abnormalities of the ex-ternal genitalia. Palpation of the testes and accessory organs will detect no dissimilar-ity from completely normal functioning glands. The ejaculate, however, may be devoid of spermatozoa, and sections of the testicular tissue may reveal marked hypo-plasia of the germinal epithelium. From this extreme to that of the normal fertile male with his full complement of millions of well shaped spermatozoa, there may be found individuals with varying degrees of deficiency demonstrating the wide range of variation common in men.[9]

Certain seminal deficiencies are due to hereditary influences, preëxisting inflam-mation, injuries, congenital malformations, and to general and local disease. Others may be the result of minor infractions of the endocrines, particularly the pituitary.

Stieve's illuminating descriptions and illustrations of the degenerative or regres-sive changes of testicular tissue in other-wise· healthy men, deserves pertinent attention.[10] Graduations of atrophy were studied, and it was shown that in the early stages there was no decrease in the size of the testicle, despite disruption of complete spermatogenesis.

This and other evidence points to the possibility that spermatogenic deficiencies may, in part, be due to incomplete elabora-tion of one or more hormones which are intimately associated with the gametogenic cells. Such an authority as Cameron has suggested, "Thus, it is easy to imagine not only the many effects which marked abnormality of the pituitary functions can cause, but also how slight pituitary changes . . . can be reflected in so many ways."

Support for this theory in the human would be forthcoming if therapeutic re-sponse more closely followed the adminis-tration of appropriate endocrine products, producing corrective changes similar to those induced in the hypophysectomized rats. In the past, several factors have obstructed a fair application of this test. The gonadotropic products offered for clinical use are standardized by biologic assay, which conveys only an approximate estimate of the strength. The unit content or available dosage has probably been far below basic requirements. It is to be re-called that a few years ago, 10 to 15 units of estrogen were used for injection in the treatment of disorders in the female. At the present time, it is customary to give several thousand units daily over a period of weeks. Collip's principle of inverse response contends that "The responsive-ness of an individual to the administered hormones varies inversely with the produc-tion of his own glands." No accurate test exists for the exact determination of the output of gonadotropic substances in the male, and accordingly, dosage has been more or less empirical and not based on estimates of need.

Despite these major handicaps, and not withstanding the many discouragements of the past, there remains the convincing animal experiments and certain isolated clinical responses which furnish a reason-able basis for continued quest and treat-ment. We have on record several well controlled cases, which have demonstrated gradual but progressive increases in sperm counts from originally insignificant num-bers to the normal millions per cubic centimeter following prolonged use of gonadotropic hormones. These have been in the distinct minority, and were unpre-dictable from case to case. Three cases are

TABLE I

SEMEN ANALYSIS

Case 1. Before Treatment	Treatment	After Treatment	Result
3/21/36 M. O. C. Withdrawal Vol. 6.1 c.c. Vis. Normal Tur. Normal pH 8.1 Motility: 1 hr. 1 motile cell per H. P. F. 3 hr. 1 motile cell per H. P. F. 12 hr. No Motility	Daily alternate injections of anterior pituitary extract 10 units and pregnancy urine extract 100 units for 106 days. Total dosage 530 units of pituitary extract and 5300 units of pregnancy urine extract.	11/11/36 M. O. C. Withdrawal Vol. 8.5 c.c. Vis. Normal Tur. Normal pH 8.1 Motility: 3 hr. 20–30 active cells per H. P. F. 16 hr. 10–12 active cells per H. P. F. 28 hr. 3–5 sluggish cells per H. P. F.	Daughter born 7/18/37
Count per c.c. 2,000,000 Total 12,200,000 Morphology: Too few cells for percentage determination. Second specimen approximately identical. Comment: Low cell count; poor motility.		Count per c.c. 8,500,000 Total 72,250,000 Morphology: Normal 87 per cent Abnormal 13 per cent Comment: Improved motility, vitality, and sperm content.	

TABLE II

SEMEN ANALYSIS

Case 11. Before Treatment	Treatment	After Treatment	Result
Date: 7/7/37 M. O. C. Withdrawal Vol. 5.5 c.c. Vis. and Tur. Reduced pH 7.8 Motility: 2 hr. 2–5 sluggish cells per H. P. F. 2 hr. 2–5 sluggish cells per H. P. F. 12 hr. No motility	8/2/37 Alternate daily injections of anterior pituitary extract 10 units and pregnancy urine 100 units for 90 days. Total dosage: 450 units of pituitary and 4500 units of pregnancy urine extracts	1/20/38 M. O. C. Withdrawal Vol. 5.0 c.c. Vis. and Tur. Reduced pH 7.9 Motility: 2 hr. 50–60 active cells per H. P. F. 6 hr. 50–60 active cells per H. P. F. 12 hr. 20–30 cells motile per H. P. F. 24 hr. 1–2 sluggish cells per H. P. F.	Wife became pregnant just before semen analysis on 1/20/38

Count per c.c. 1,800,000
Total 9,900,000
Morphology: Too sparse for percentage count
Second specimen confirmed above
Comment: Oligospermia
    Poor motility
		Count per c.c. 51,000,000
		Total 255,000,000
		Morphology: Normal 91.5 per cent
		    Abnormal 8.5 per cent
		
		Comment: Improved motility vitality, and sperm count.	

presented here in abstract form to illustrate such favorable response to hormonal therapy.

Case i.   J. B., age 35, was seen March 31, 1936. His marriage was barren, although two siblings had children. He had had mumps at age 30, with the right testicle enlarged for three to four days. He had been married for

This patient had been married three years, having coitus two to three times a week. He had used contraceptive measures until ten months previous.

He was well developed and proportioned with normal genitalia. His wife, according to gynecological reports, had no demonstrable cause for infertility. Urine and Wassermann tests were negative. The basal metabolic rate

TABLE III
SEMEN ANALYSIS

Case iii. Before Treatment	Treatment	After Treatment	Result
Date: 6/7/37 M. O. C. Withdrawal Vol. 0.4 c.c. Vis. & Tur. Reduced pH 8.0 Motility: 1½ hr. 3–8 sluggish cells per H. P. F. 6 hr. very occasional cell motile	9/15/37 to 11/9/37 Alternate injections of anterior pituitary extract 100 units and pregnancy urine extract 500 units for 20 injections. Total dosage was 1000 units of pituitary extract and 5000 units of pregnancy urine extract.	12/23/37 M. O. C. Withdrawal Vol. 1.6 c.c. Vis. & Tur. Reduced pH 8.1 Motility: 1 hr. 80–120 active cells per H. P. F. 6 hr. 80 to 100 active cells per H. P. F. 12 hr. 20–40 active cells per H. P. F. 24 hr. 4–6 sluggish cells per H. P. F.	No pregnancy reported. Check on specimen 4/15/38 was essentially same as 12/23/37.
Count per c.c. 6,000,000 Total 2,400,000 Morphology: Too sparse for percentage count Comment: Oligospermia Poor motility		Count per c.c. 73,000,000 Total 116,800,000 Morphology: Normal 82 per cent Abnormal 18 per cent Comment: Decided improvement. Relatively high percentage of abnormal forms.	

five years, had used contraceptives the first two years, but none for the past three. In spite of coitus one to two times a week, there were no signs of pregnancy.

The patient was a well developed male. General physical examination was negative. The testes, epididymes, vasa and prostate were essentially normal.

A gynecologist's report showed no reason on the wife's part to explain the infertility.

The urine and Wassermann were negative, the basal metabolic rate minus 10 per cent. The semen analysis and its reaction to treatment are indicated in Table i.

Case ii.   T. B., age 44, was seen June 23, 1937. He had had no serious illnesses, operations or venereal infection, but had had a chronic prostatis which was treated five years before. Of his three sisters, only one had married, and she had one child.

was plus 7 per cent. Semen analysis and its change under treatment are indicated in Table ii.

New preparations are now offered which are five and ten times as powerful as the old ones. Cases now being treated with these refined gonadotropic principles have in some instances shown some unusual changes, not encountered during the past few years. A report at this time is hardly justified, because of the short period of observation and relatively few cases under the newer treatment. The following case is presented, however, with the full realization of this fact.

Case iii.   J. C. B., age 35, was seen June 4, 1937 because of possible miscarriages. He had been married two years, had used no contra-

ceptives for one year. Coitus occurred one to three times a week. Two periods had been delayed one month, which brought up the possibility of miscarriages.

The patient had had no serious illnesses, no operations or venereal infection. Of his four siblings, all but one had children.

The patient was somewhat obese, but the general physical examination proved otherwise negative. Both testicles were high in the scrotum and slightly smaller than normal. The prostate was normal. Urine was negative, as was the Wassermann test. The basal metabolic rate was minus 11 per cent. The response to treatment is indicated in Table III.

As knowledge progresses and as endocrine products are strengthened, standardized and fractionalized, it seems reasonable to believe that rational and effective therapy may evolve which will offer consistent results to certain men with impaired reproductive capacities.

SUMMARY

1. The basophilic cells of the anterior pituitary gland are intimately concerned with elaboration of gonadotropic hormones.

2. Two fractions of gonadotropic hormones are recognized: the follicle stimulating and the luteinizing fractions. These act on the interstitial and spermatogenic cells of the testes.

3. There are three principal sources of gonadotropic hormones in the normal human: (a) anterior pituitary extract; (b) pregnancy urine; (c) castrate urine.

4. All of these have varying amounts of the two fractions and act accordingly.

5. Biologic activity of these hormones is substantiated by animal experiments and human studies.

6. Androgens are derived from the gonads and are now synthetically prepared.

7. Androgens effect secondary sexual characteristics and genital structures, but probably have little action on human spermatogenic tissue.

8. Anti-hormone response may be a foreign protein immunity.

9. Minor endocrine disturbances may account for a certain percentage of seminal deficiencies.

10. Problems of treatment are discussed.

11. Three cases are presented illustrating improvement, coincidental to use of the gonadotropic principles.

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THE RADIATION TREATMENT OF CANCER OF THE BREAST

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THERE has been so much written on the use of radiation in the treatment of cancer of the breast that any additional report must have a special purpose. The following study of breast cancer is the result of years of personal observation of a group of rather unusual cases encountered at the Radiological Institute of the University of Istanbul, Turkey. Many of these patients were quite young; most of them did not seek medical advice until a relatively advanced stage of the disease. The incidence of local postoperative recurrence was unusually high, and in many of the patients cutaneous metastasis of a lymphangitic type developed. These and other peculiarities gave rise to a wealth of problems for discussion and decision and influenced our treatment considerably. The experience derived from this somewhat unusual group of cases, though not readily transferable to conditions elsewhere, may be of considerable significance to physicians interested in breast cancer.

At the present time there are two main therapeutic approaches to the treatment of carcinoma of the breast; surgery and radiotherapy with either Roentgen rays or radium. As for the surgical treatment, provided indications for surgery exist, there is fairly universal agreement as to the procedure to be employed, although—as will be shown later—even this question is not infallibly settled. The question of radiation treatment, on the other hand, is by no means clearly defined, either as to the technique or indications. There is considerable difference of opinion as to whether or not preoperative radiation should be given; as well as the appropriate interval before operation and technique of adminis-

tration. Similarly, there is diversity of opinion concerning the time, intensity, duration and technique of postoperative radiation, to say nothing of additional radiation for sterilization of the younger women in the child-bearing age. These questions have not been clearly answered and deserve some discussion and clarification.

The number of patients whose symptoms and therapeutic problems constitute the basis for this report is rather small, amounting to only sixty cases. However, a continuous three year period of close personal observation and the multiplicity of symptoms and findings make this clinical material of some value. Furthermore, similar observations have been made on a far larger group of cases in the past two years; however, the latter cases are not included in this study as the period of observation is thought to be insufficient for this report.

Most of these patients came from very poor surroundings in a rather sparsely populated country, where the quality of medical attention (except for the few larger cities) cannot be compared to that of Western Europe or North America; diagnostic and therapeutic facilities in Turkey are still quite limited and women do not seek medical attention there until they are in desperate need of it. Furthermore, the older women of cancer age still suffer from traditional religious superstitions (which the younger women have partially overcome) leading to an aversion towards being examined by men.

With such conditions prevailing, it is not surprising that only four of these sixty women suffering from cancer of the breast presented themselves for first examination while the tumor was still localized, without

extension to skin and without metastasis. (Such early cases of breast cancer without skin involvement or metastatic glands comprise the so-called group I, according to the Steinthal classification.) Thirty patients had axillary lymph gland metastasis and some degree of fixation of the tumor to the skin (attributes of group II in the Steinthal grading) at the time of their first examination. Twenty-one others showed advanced local extension of the growth, axillary and supraclavicular lymph gland metastases (Steinthal III) and five others presented themselves for the first time with extensive distant metastases, in hopelessly advanced condition. The exact clinical picture, the extent and severity of the disease in these patients is not sufficiently clear from this brief description, based on Steinthal's classification. It may help to mention that of these sixty patients twenty-one had multiple skin metastases, which in a few of the cases consisted of small nodules in the mammary lymphatics, but in the majority had progressed to generalized stony hard infiltration of the entire breast and frequently to cancer en cuirasse.

To facilitate the interpretation of the findings and conclusions to be presented later, the course of the disease in these sixty cases is described briefly in the following:

Steinthal I: Four patients, operated on by radical mastectomy and then given postoperative radiation. In one of these cases there was a period of one and three-fourths years between operation and radiation during which interval cerebral metastases had already developed. This patient died of the metastases before radiation could be completed. The other three patients are symptom-free.

Steinthal II: Thirty patients, twenty-eight of whom had been operated on previously. Two patients had refused operation, and later, when they were willing to submit to it, had developed extensive skin infiltration rendering surgery impossible. Of these thirty women, twelve presented themselves for treatment with recurrences in the operative scar, nine with extensive skin metastasis. In most of these cases radiation had not been given following operation. At the start of radiotherapy in our department metastases were present as described. Seven patients, who received radiation therapy soon after operation (up to six weeks) are free of disease.

Steinthal III: Twenty-one patients, nineteen of whom underwent operation, four following previous radiation. Twelve of these women, who had been operated on, returned for treatment with local recurrences in the operative field, ten of these with extensive skin metastases. Sixteen of the patients had metastases in other organs than axilla and supraclavicular fossae. Of these twenty-one women, one is now free of all evidence of disease three years after treatment; in four other cases the primary tumor and metastatic adenopathy regressed sufficiently under radiation treatment to permit later operation. These four patients are now well even though the apparent cure may not prove to be permanent. In the remaining patients, following transitory improvement and temporary comfort, recurrence developed; some have died of metastases; others have not returned to the clinic and no further information can be given as to their outcome, which we must assume to be unfavorable. Of the remaining five patients who came for treatment with distant metastases already present, two did not return to the clinic, the remaining three died in two months, eleven months and one and one-half years after the beginning of treatment.

In reviewing this summary of results, certain features stand out: first, the fact that so few of the patients came in an early operable stage of the disease, second, that so many of the cases were operated on in stage III of the disease (Steinthal), the prognosis of which is very poor following surgery. In most of these cases radical operation was not possible and palliative procedures were carried out because many of these patients could not receive radiation

therapy due to the lack of facilities for such treatment; in addition, radiation was not infrequently discouraged by surgeons who were not convinced of its value. Furthermore, when radiation was accepted or recommended, a prolonged interval had usually transpired since operation. Very few of our cases were referred directly from the surgical clinic following the healing of the operative wounds; in most cases many months had passed before the surgeons advised radiation and their decision in general depended on the appearance of local recurrences or metastases. It is obvious that under such conditions the prognosis following radiation treatment must inevitably be bad. Furthermore, many of the patients did not come to the radiation institute for years after the operation, either because their physicians had regarded radiotherapy with doubt and suspicion or had actually discouraged it.

A striking feature of this group of cases is the unusually large proportion of patients with skin metastases, which had progressed to cancer en cuirasse in six women and in fifteen others had produced widespread nodular infiltration of large areas of skin. The treatment of such cutaneous and other metastases with their attendant severe symptoms (pain, limitation of motion and function, shortness of breath) constituted an important part of our work. The therapy was carried out in various ways and with varying results; the selection of the method or technique was often limited by the means at our disposal (nearly all patients were treated free) and the treatment could not always be administered under ideal medical conditions. Without going into the details of our therapy, it might be mentioned that our best results were obtained with interstitial radium and Roentgen rays at close focal distance to the skin, using the so-called concentrated fractionated method.

Naturally, we wondered why this particular group of cases of breast cancer should exhibit such a large proportion of cutaneous metastases (more than one-

third of all our cases). While this question is difficult to answer, certain factors may have been of contributory importance. In the first place, our patients were often very young; in the sixty cases, three were under 30, eighteen under 40, and seventeen between 40 and 50 years of age. Skin metastases were especially frequent in the younger patients. In addition, the type and manner of execution of the surgical procedure may have been of importance; a large percentage of our patients were operated on even when the prognosis following such a procedure was expected to be inauspicious. Careful review of the histories, however, revealed that most of our patients with skin metastases had been operated on in the best clinics of the country (or of Istanbul) and by the outstanding surgeons; so that it is difficult to attribute such recurrences to the surgical procedure. Furthermore, it is of significance that most of the patients came for radiation therapy rather late, often too late. Finally, one wonders whether these patients with such extensive cutaneous involvement had special types of breast cancer predisposing to this variety of metastasis. From histologic study of those cases in which specimens were obtained, we could find no explanation for such tendency to cutaneous metastasis.

In the absence of a more definite explanation for such skin metastasis it may be assumed that delay or omission of radiotherapy, or its inadequate execution, may at least have been of some significance. The low standard of living conditions compared to those of Western Europe and North America probably had little to do with the prognosis in these cases; for in all patients suffering from cancer in general (including breast) in this region, we have noted relatively good resistance towards the disease, as manifested by good general condition despite widespread involvement of the body by cancer, infrequency of cachexia and the possibility of treating a large proportion of our far advanced cases as ambulatory patients.

The same factors contributing to the frequent occurrence of cutaneous metastasis in our cases of breast cancer are also responsible for our poor results in the treatment of this condition compared to those of other authors. Since the alteration of the unsatisfactory conditions depended on gradual education of the physicians and lay population, we forced ourselves to undertake the treatment of such advanced cases despite unfavorable circumstances and tried to improve the results in the radiation treatment of this desolate group of cases by variations in technique. The details of such treatment are of interest only to radiologists, but it is obvious from the brief review given above that our efforts were not entirely futile.

We are inclined to attribute the good results in the treatment of the early cases of breast cancer (Steinthal I) to radical operation, but the value of radiation even for such cases should not be minimized. The importance of radiation is indicated by the one of our four stage I cases that developed cerebral metastases, one and three-quarter years postoperatively (this being the only stage I case that did not receive postoperative radiotherapy soon after operation). It might have been possible to avert such metastases and rapidly fatal result by early radiation. We have since observed a series of similar patients with localized breast tumors subjected to surgery but not receiving postoperative radiation and returning one to two years later with extensive distant metastases; some of these cases occurred in younger women in conjunction with pregnancy.

Of thirty women referred for radiation treatment in stage II (Steinthal), we know seven to be free of all evidence of disease. In these seven cases the importance of radiation is unquestionable; for these cases were all irradiated soon postoperatively (two to six weeks) and further spread and recurrence were doubtless avoided.

The results obtained in cases irradiated in stage III (Steinthal) are of special interest. Of twenty-one patients, four were so improved by radiation that operation became possible; following additional radiation given postoperatively the supraclavicular glands disappeared and these four patients are now free of all evidence of disease three years following operation. A fifth patient has been kept symptom-free by repeated radiation. In several other cases it was not possible to cure the primary tumor or glandular metastases but it was possible to so help these women that they continued to live useful lives for considerable periods. One feature of these far advanced cases seems worth mentioning. To our surprise we observed that the cases previously operated on by radical mastectomy at the larger clinics were either not at all or very little helped by radiation. Not only was it impossible to obtain curative results or even slight improvement despite large doses of radiation but not infrequently distant metastases appeared during radiation. In contrast to these cases, the women who had been subjected to less adequate surgery in the smaller hospitals, with only partial removal of the cancerous tissue, often reacted very well to radiation treatment.

Since this peculiar reaction of previously operated breast cancer to radiation was observed in about one-third of our patients, this question becomes rather important. While it is unsafe to draw general conclusions from these cases with rather uncommon course of events, it is important to watch for similar behavior in other cases of breast cancer. At least in our series, the cases inadequately operated on (from the surgical point of view) reacted more favorably to radiation than those subjected to radical mastectomy. Two factors may contribute to this type of response: on the one hand, the danger of disseminating the disease may be greater with the more radical surgical procedure; secondly, the removal of larger masses of tissue may impair the healing capacities of the remaining tissue. In the execution of radiation treatment the protection of the normal

tissue surrounding the tumor, which is largely responsible for healing, is as important as the destructive effect of the rays on the tumor. If no such surrounding tissue is present, or if it is so damaged by previous measures that its capacity for recovery is impaired, a radiation treatment which depends in large part on such healthy reactive tissue can not be accomplished successfully. The observations mentioned above seem to confirm such impressions.

It is difficult to draw general conclusions from these advanced and often truly desolate cases of breast cancer; in other countries, women suffering from breast cancer usually come for treatment at far earlier stages of the disease and the results of treatment are apt to be more favorable. The fact that in spite of the advanced condition of our cases and other handicaps, fairly good results were occasionally obtained, particularly in the very advanced cases, is convincing proof of the effectiveness of systematically executed radiation treatment. On the basis of our experience, which corresponds to that of many others in the literature, there is no one method of treatment applicable to all cases of breast carcinoma, and in various stages of the disease either surgery or radiation is to be preferred. The selection of the method for any specific case can be made only after a thorough study of the general condition of the patient and of the special pathology. There are, however, certain general principles applicable to the treatment of breast cancer; these may be summarized as follows:

1. For tumors localized to the breast, without attachment to skin or underlying tissues, without demonstrable glandular metastases, radical mastectomy is indicated. In such cases postoperative radiation should be administered to the operative field and sites of regional gland metastasis; the radiation should be repeated in three months. In all cases in which the presence of glandular metastases is at all questionable, preoperative radiation is indicated, followed by radical mastectomy. The operation is in no way affected by such radiation and the prognosis is improved.

2. In all cases of breast cancer with skin attachment and axillary metastasis, preoperative radiation is indispensable. In many cases the tumor and axillary glands will be diminished in size and not infrequently all evidence of disease will disappear. Operation is never aggravated but is always facilitated by such radiation. Following operation, radiation should be administered and should later be repeated depending on the individual case. With such a plan of treatment it will be possible to perform radical surgery in most cases.

3. All other cases in which the tumor is already attached to the underlying tissues and in which supraclavicular glands are present should not be operated on. In this group of cases, radiation is often of great use and not infrequently leads to a disappearance of the tumor mass and the metastatic glands. Following radiation, or sometimes during its execution, it may be helpful to utilize surgery either for removal of portions of the tumor or for more complete operation if the disease has regressed sufficiently to warrant such procedure. On the basis of our experience we would advise against extensive surgical procedures in advanced cases for they seldom give good results following such surgery and the outlook following later radiation is unfavorably influenced. This statement may be questioned by surgeons, for at first glance it seems to contradict the principles of surgery. However, many surgeons advise against extensive operative procedures in such advanced cases, and in these cases the radio-therapist should be largely responsible for the entire management of the case and the recommendation of any special procedures to be carried out. The removal of large amounts of tissue should be avoided if a better result is to be anticipated by radiation in advanced cases of breast cancer.

There is an additional variety of radiation treatment which deserves special

mention and which seems indicated, on the basis of our experience. This is the sterilization by radiation of young women in the child-bearing age suffering from breast cancer. We have had the opportunity of treating an exceptionally large number of young patients with carcinoma of the breast, more than half of these patients not having undergone the menopause. We have already mentioned the apparent increased tendency to metastasis in the younger patients and the relatively malignant course of the disease in such women. In other cases, we have observed the coincidence of pregnancy and local recurrence or *metastases. All these considerations have* led us to sterilize by radiation all women developing cancer of the breast prior to the menopause. We have observed no unfavorable effects from this procedure and in many cases have noted a remarkable improvement in results. This procedure may constitute prophylaxis against an otherwise unfavorable outcome.

We shall not discuss here the questions of special interest to the radiotherapist, such as the time, the intensity and quality of the rays and the size of the dose as affecting the therapeutic result. Similarly, we shall not consider here the relative indications for radium or x-rays which may be important for the result in the individual case. Each case presents special problems which can be decided only by collaboration between internist, surgeon and radiotherapist. Nevertheless, the principles suggested for the treatment of breast cancer in general seem rational. If our experiences may prove of value to others in the management of cases of breast cancer the purpose of this article has been fulfilled.

SUMMARY

The results of radiation treatment for cancer of the breast in a rather unusual group of cases observed in Turkey are discussed. A large proportion of these cases of breast cancer occurred in rather young *women most of whom first presented them-*selves for examination or treatment in a very advanced stage of the disease. Furthermore, many of the patients developed postoperative recurrences and an unusually high percentage developed lymphatic cutaneous metastases.

The selection of the method of treatment to be employed in the individual case depends on the general condition of the patient, the stage of the local pathology and an accurate evaluation of the effectiveness of the various possible methods of treatment from the viewpoint of the general practitioner, surgeon and radiologist. These points of view are presented with special consideration of the indications for radiotherapy. Sterilization of women in child-bearing age suffering from cancer of the breast is recommended.

EXTERNAL LATERAL DISLOCATION OF THE ELBOW*

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DISLOCATIONS and fractures in or about the elbow are frequent and, because of the commonly resulting deformities and disabilities, are a constant challenge to the surgeon. In spite of accumulated experience and a wealth of literature, many problems, involved in these injuries, remain to be solved. Having encountered, within a short time, several examples of a rather unusual type of elbow dislocation, it seems that a study and review of that type might be justified.

It is the purpose of this article to present the results of a study of external lateral dislocation of the elbow, with case reports. Dislocation of both bones at the elbow can occur in any one of four or more directions, i.e., anterior, posterior, lateral, medial, and divergent. There are, of course, variations in these general types. There seems to have been some confusion in describing lateral dislocations, some writers considering the displacement of the humerus as the basis for distinction. In this discussion, the direction in which the ulna and radius are displaced is used to differentiate between the lesions.

Dislocations are probably the most frequent of elbow injuries, exceeding fractures in most series; in one of these they constituted 20 per cent of the cases.[1] The simple posterior dislocation is the most frequent; this is followed in order by the posterior with fracture; dislocation of the head of the radius; internal lateral, or medial; and the external lateral type. In a study of a series of dislocations by Geist and Henry,[2] external lateral dislocations occurred in only 0.7 per cent. A review of other studies also shows that the external lateral type is rather rare. Newell,[3] in a series of 1,114 cases of elbow injuries, records twenty-one dislocations none of which were lateral. The case reports of this variety of dislocation, as revealed by a fairly extensive review of the literature, are appended.

External lateral dislocations of the elbow may be either partial or complete. Tinker[4] stated, "Partial outward dislocation of the elbow joint is so unusual that every case seems worth recording." He reported two cases of partial dislocation, one of which was compound. Stimson,[7] in discussing external lateral dislocation, referred to only thirteen cases of partial dislocation, including one of his own, and believed that this represented the entire number reported up to that time. Concerning complete outward dislocation, he states, "These dislocations, of which the first observation was reported by Dupuytren in 1807, although the form had been described by Petit nearly one hundred years before, were apparently so rare that Malgaigne could collect only ten reported cases. Of late, reports have so multiplied that, excluding irregular cases, those of which the description is not sufficiently detailed and those which seem more properly to belong among dislocations backward, the number of those available for study and generalization is about twenty-five." Wilson[1] reported three lateral dislocations in a series of ninety-three patients with dislocations, not indicating the type. Armitage and Armitage[5] state, "External lateral dislocation of both bones of the forearm is very rare."

Winslow[6] in reporting his only case of complete external lateral dislocation describes three varieties. He makes the following statement: "Owing to the anatomic
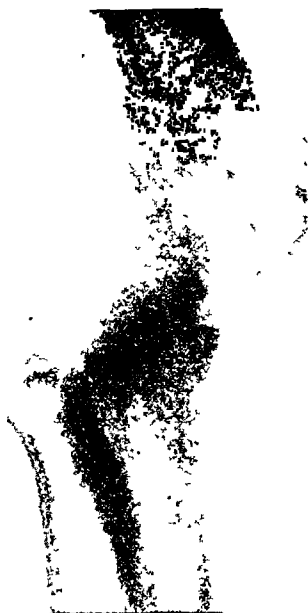
FIG. 1. Case 1. Anteroposterior view of incomplete external lateral dislocation. (August 5, 1935.)

FIG. 2. Same case, showing detached and displaced portion of epitrochlea.

FIG. 3A. Case 1. Anteroposterior view after reduction, showing fragment of epitrochlea in proper position. (October 29, 1935.)

FIG. 3B. Same case, showing complete reduction.

complexity of the elbow joint, complete lateral dislocations are of extreme rarity." His classification is as follows:

1. Directly outward, without rotation of forearm.

2. Subepicondylar, with elbow flexed nearly or entirely to a right angle with forearm pronated, the radius being placed somewhat anteriorly to the ulna. The great sigmoid cavity is located just below the external condyle.

3. Supra-epicondylar, the forearm being flexed in pronation. The bones of the forearm are placed above the external epicondyle, with considerable shortening of the arm, which constitutes the most marked type.

Mechanism. A study of this injury indicates that it is produced by a very unusual degree or type of violence. It apparently results in most cases from a fall on the outstretched hand, although it is said to have followed a fall on the inner side of the elbow, or from a direct blow upon the forearm. Tinker[4] believed that this dislocation scarcely ever occurs in a normal adult. He further stated that it occurs only in

Nicoladoni quoted by Tinker, believed that in most cases, evulsion of the internal condyle or epitrochlea in these dislocations

Fig. 4. Lateral view in same case after reduction.

Fig. 5. Case II. Anteroposterior view, showing complete external lateral dislocation with radius and ulna impinged on the supinator ridge. (August 12, 1935.)
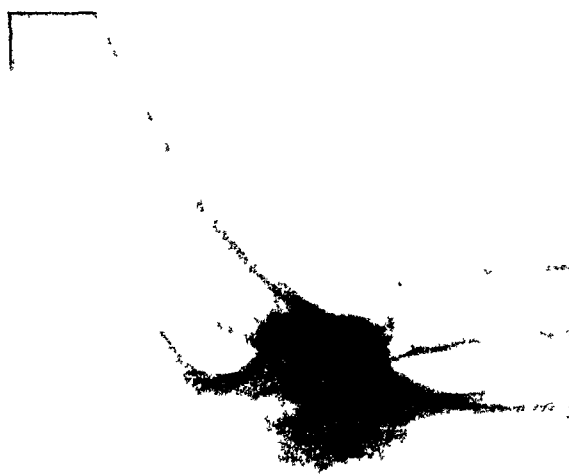
Fig. 6. Lateral view of same case, showing displacement of both bones and fracture at upper end of the radius.

persons who have an abnormal weakness of the capsule and internal lateral ligament of the elbow joint. This no doubt accounts for the infrequency of the lesion.

is caused through the tension on the attached flexor muscles. In children, this is probably an epiphyseal separation. Tinker attributed this condition to the pull on the

internal lateral ligament. He produced the dislocation experimentally by abduction of the completely extended and pronated

FIG. 7. Case II, immediately after reduction, showing improved position of head of radius. (August 12, 1935.)

forearm. Rotation on the humeroradial articulation as a center causes the rupture of the internal lateral ligament, thus

of the trochlea, sliding over the outer ridge of this process, occupying a position varying from the groove between this ridge and the capitellum, in the incomplete form, to a position upon the supinator ridge or epicondyle, in the complete form.

Physical Signs. Examination usually shows the forearm in a position between extension and right angle flexion, with a variance of angulation depending on the individual case. Pronation of the forearm is common and is due to pressure under the muscles which take attachment from the external condyle and are made tense. When the hand of the injured extremity hangs at the side, with the forearm adducted, the axis of the humerus is pointed downward and inward. The normal angle of the humerus is more downward and outward. The nternal condyle is made prominent, the skin being made very tense over it and it may have a rough edge if the epitrochlea has been avulsed. There is increased protuberance of the oleocranon, because the ridge of the sigmoid cavity interlocks in the deep groove between the outer margin of the trochlear surface and the

FIG. 8. Case III. Anteroposterior view, showing partial external lateral dislocation, with displacement of epitrochlea downward and backward. (August 11, 1935.)

FIG. 9. Lateral view in same case, showing displaced epitrochlea.

opening the joint. The sigmoid cavity of the ulna is then separated from the trochlea and its mid-ridge escapes from the groove

capitellum. The external condyle is usually not prominent, but may be identified by making strong pressure above the head of

the radius, behind the extensor muscles. The very definite widening of the transverse diameter of the joint is the most prominent sign. The triceps tendon stands out as a very tense band.

Complications. These dislocations may be compound and complicated by severe injuries to soft and osseous tissues. Extensive laceration of the capsule and overlying soft tissues, particularly the musculature, may occur. The chief vessels and nerve trunks may be seriously injured, although this complication is not frequent in the literature. Jeanneney and Viella[9] cite a case in which such trauma occurred and was followed by the Volkmann syndrome. Juvara, in 1930, quoted by Papin,[10] reported a case in which there was a paralysis of the cubital nerve. These complications of the soft parts may be so extensive as to require amputation. Gant,[11] in 1866, reported a case of external latero-angular dislocation in which this operation became imperative on account of the extensive damage to the soft parts. Gerdes,[8] 1863, reported a case of complete external

FIG. 10. Case III, showing reduction, with epitrochlea still displaced, but in improved position. (September 8, 1935.)

FIG 11A Case IV. Anterior-posterior view, showing partial external lateral dislocation, with evidence of disturbance of external condyle and displacement of epitrochlea into olecranon fossa of humerus. (October 28, 1935.)
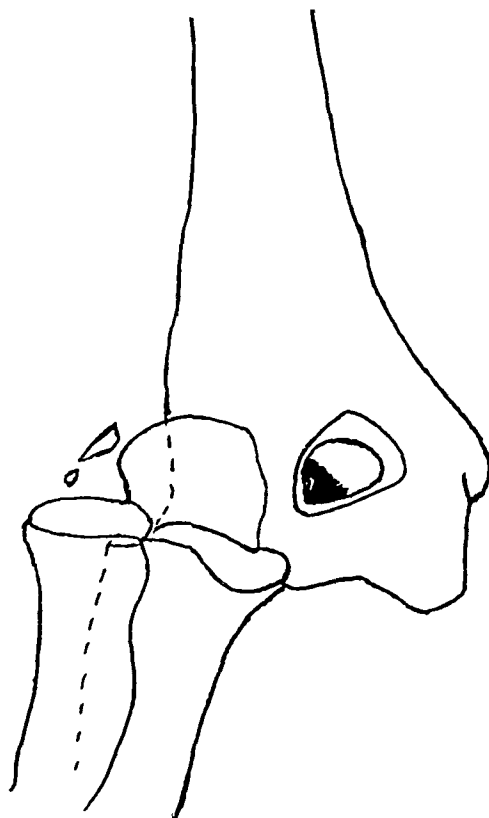
FIG. 11B. Diagrammatic representation of Case IV.

lateral dislocation, with such extensive trauma to the soft parts, that, in spite of non-injury to chief vessels and nerve

FIG. 12. Case IV. Lateral view, showing epitrochlea displaced into olecranon-fossa of humerus.

trunks, removal of the limb was deemed necessary. These amputations probably might have been avoided by the more modern débridement and aseptic technique.

The internal condyle or epitrochlea is frequently fractured or avulsed and may become interposed in the disturbed joint. This condition increases the difficulties of both diagnosis and treatment. The location of the fragment may cause an external appearance of the parts, simulating fracture of the olecranon (Papin[10]). The external condyle may also be traumatized, although this does not seem to increase the difficulty of reduction. In addition to fractures in or about the elbow joint, there may be such injuries at the lower levels of the forearm, Colles' fractures probably being the most frequent. The additional fractures are no doubt due to the unusual type and degree of violence necessary to produce this dislocation.

Diagnosis. Prompt and accurate diagnosis of injuries involving the elbow region is urgent, for upon it depends the selection
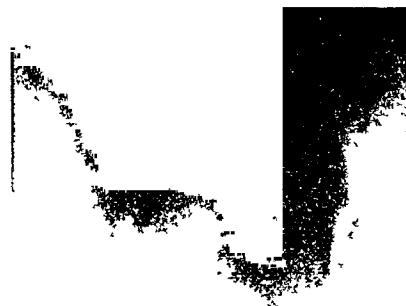
FIG. 13. Case IV. Anteroposterior view after reduction. In flexion. Showing replacement of epitrochlea and indicating damage to external condyle. (October 28, 1935.)

of the method of treatment. Treatment must vary with the kind of injury. Favorable prognosis is contingent upon adequate treatment. These are pertinent factors in caring for the dislocation under discussion. The correct diagnosis may be very evident from the physical signs, but careful x-ray examination should be made to establish a more accurate presentation of the conditions involved. The whole forearm should be included in this study, especially when there is evidence of gross injury in the elbow region. In two of the cases to be reported, Colles' fracture was present and added to the difficulties of treatment and prognosis.

Treatment. While our experience in the treatment of these external lateral dislocations is limited, certain points gathered from it and from a review of the reported case histories seem worthy of consideration. Injuries to the soft parts, especially to the chief nerve trunks and vessels, should receive careful attention before any manipulation is undertaken. In some cases, these injuries may be more important than the underlying fracture or dislocation. As these complications are not peculiar to the dislocation under discussion, their manage-

ment will not be further elaborated upon. Attempt at reduction should not be delayed, for early reposition must be obtained should be made promptly. This can be accomplished in the operating room before splinting. Hospitalization of such cases for

FIG. 14. Case IV. Anteroposterior view, showing evidence of union of epitrochlea to humerus and reparative process of external condyle with thickening of adjacent periosteum. (December 2, 1935.)

FIG. 15. Same case, showing further union of epitrochlea. (March 9, 1936.)

to secure a favorable end result, and also, because reduction becomes more difficult if delayed. Older cases should have at least one attempt at closed reduction. Those eight to ten days old are difficult and are rarely reduced without operation.

General anesthesia to obtain complete relaxation is usually required, for without it effective manipulation is painful and difficult because of muscle spasm. Reduction will usually be obtained by manipulating the forearm into strong pronation, at the same time extending it and continuing with lateral flexion and supination. After reposition, the arm is put into acute flexion, depending in degree upon the severity of the swelling about the joint. Early reduction often obviates the difficulty of swelling. These cases are usually retained by a posterior moulded plaster splint, extending from mid-humerus to the distal ends of the metacarpals, with the forearm in supination. An x-ray check

several days is a wise precaution in order to facilitate observation of peripheral nerve function and circulation.

Reduction may be comparatively easy and perhaps may not require any prescribed technique other than straight traction, followed by flexion. However, certain cases may be resistant, the cause of this resistance not being evident in the x-ray plates. In these, continued or repeated forceful attempts at reduction should not be made for fear of added damage to the soft parts, especially nerves and vessels that may be atheromatous.

If closed reduction fails in competent hands, we agree with Winslow[6] and Papin[10] that open operation should be done at once, for, while there is risk of infection in open reduction, the importance of obtaining prompt reposition is so great that, under proper conditions, it seems worthwhile to assume this risk. Cases showing interposition of the displaced epitrochlea or other

fragments indicate early surgical inter-
ference, if manipulation does not attain
proper reposition. An interposed epitroch-
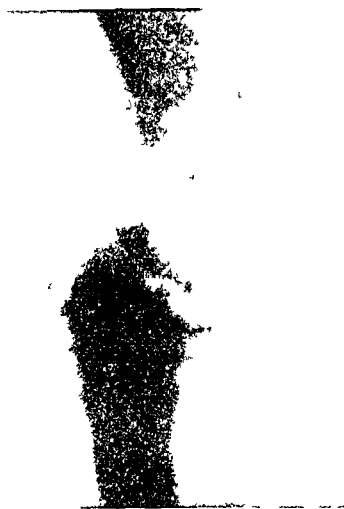
FIG. 16 Case v. Anteroposterior view
showing complete external lateral dis-
location in subepicondylar position.
(September 30, 1935.)

lea should be dislodged and the elbow
reduced; then if large, it may be fixed at
its proper position; if small, it may be
removed entirely. In one case, not person-
ally treated but reported with this paper,
open operation was done three weeks after
the injury, but no attempt was made to
replace the epitrochlea. Postoperative x-ray
showed that the reduction had been main-
tained but with a fair-sized epitrochlea still
widely displaced, with no interposition.
This condition apparently has not inter-
fered with the end result. However, we
believe it wise to deal definitely with this
process of bone, even if it is not interposed.
Guerin, quoted by Papin,[10] cites a case in
which the epitrochlea came out of the
articulation, anteriorly. By means of a
bodkin thrust through the skin, he replaced
the epitrochlea into position.

Open operation affords an opportunity
for inspecting the joint with respect to
disturbing elements such as interposed
tissues or small bone fragments, which may
have been missed in the x-ray picture. It
allows for the careful removal of blood
clots that might later undergo osseous

changes and disturb joint function. This
inspection of the joint cavity must be most
careful, with a minimum of manipulation,
and should never include digital examina-
tion. In one case presented, in which closed
reduction failed, early operation revealed
conditions which would have resisted any
further manipulations in attempting closed
reduction, and which no doubt would have
resulted in further damage to soft parts.
Unreduced cases will usually result in
disabled and painful joints, requiring
radical treatment later. Occasionally an
old unreduced case may have a degree of
function which does not warrant any
attempt at improvement.

If there is much skin damage or swelling
about the joint, operation must be delayed.
However, this is a matter of circumstance
and judgment. We feel that under proper
conditions, patients seen within three hours
after the accident and considered operative,
may be operated upon at once with com-
parative safety, for within this period it is
believed that damaged tissues retain a
definite resistance to infection.

Active motion, with arm supported in
splint should be started early—within
twenty-four hours in cases treated by
closed reduction and in thirty-six hours in
cases which have received operative treat-
ment. At first this active motion must be
carefully guarded and be limited to flexion,
depending upon the degree of pain and
swelling. Under strict supervision, prona-
tion and supination with flexion should
follow, so that within three or four weeks
satisfactory function should be attained in
most uncomplicated cases. The more com-
plicated cases will also show a better
percentage of good results, if early, properly
supervised active motion is instituted.
Proper physiotherapy is also an aid to
recovery, but should not be depended upon
to the exclusion of the patient's own
motion. One of the causes of unsatisfactory
results in elbow joint cases has been pro-
longed immobilization. Some of the earlier
writers recognized this factor which has
been too much overlooked in later years.

F. M. Johnson,[12] in 1880, called attention to the danger of fixing joints for too long a period, citing an elbow case with resulting

FIG. 17. Case v. Lateral view, showing lateral displacement with a splinter fracture of the ulna. (September 30, 1935.)

ankylosis from such a procedure. He advocated passive motion. However, we feel that careful active motion is the better method. H. J. Bigelow,[13] in 1868, spoke of the "general impropriety of passive motion in treatment of fractures and dislocations of the elbow joint."

Prognosis. Prognosis must always be guarded in these cases as in any injury involving the elbow joint. Even though complete and early reduction has been obtained in a simple case, secondary joint changes may take place and cause more or less disability. The causes of these disabling alterations have not been entirely explained. Certain writers believe that, because of the numerous centers of ossification in the elbow region, an injury involving them may cause irritation with resulting excessive ossifying changes in the fracture lines, as well as ossification of blood clots and damaged muscle tissues. Such changes may occur in the absence of demonstrated fracture lines.

These cases should have adequate follow-up treatment and observation and be finally rated as to result at the end of a year. This procedure is not always possible

and end results cannot always be ascertained. However, we believe that, if early reduction is accomplished in these disloca-

FIG. 18. Same case, showing reduction, with slight upward displacement of fragment of ulna. (October 1, 1935.)

tions, whether simple or complicated, the results will be much improved.

CASE REPORTS

CASE I. A. G., aged 10 years, entered the hospital August 5, 1935 at 8 P.M., immediately after a fall on his outstretched hand. Examination showed a deformity of the right elbow with marked widening. The forearm was held in extension, with slight pronation. The diagnosis was an unusual dislocation of the elbow, with possible fracture. There was no disturbance of the peripheral circulation or nerve function. X-ray examination (Figs. 1–4) showed an incomplete external lateral dislocation of the elbow joint, with avulsion of the epitrochlea.

Treatment. Under general anesthesia, an attempt at reduction was made immediately. This was unsuccessful and, as further effort seemed useless, it was abandoned for fear of causing added injury to the soft parts. The cause of failure was not evident on further examination or in a study of the x-ray plates. Consent for immediate operation could not be obtained. The patient was put to bed, with forearm in traction; this treatment was maintained for twelve hours. It was hoped that this traction might overcome the muscle spasm and perhaps allow reduction. A second effort to reduce was unsuccessful and permission for

operation was given. The operation was performed about thirteen hours after the accident. A posterolateral incision exposed the interior

FIG. 19. Case v. Showing complete reduction. (October 1, 1935.)

of the joint, revealing a situation that would have defied any attempt at closed reduction. The radius was displaced anteriorly with its head forced upward through the orbicular ligament and the anterior portion of the joint capsule. It was firmly fixed in this position by a constricting band around it, immediately below the head. Before reduction could be accomplished, this band required incision. The joint was then carefully flushed with normal saline solution, removing a moderate amount of partially clotted blood. After reduction of the joint and replacement of the epitrochlea, the torn orbicular ligament, joint capsule and other injured tissues were repaired with catgut. The skin was closed with silkworm gut tied over small rubber tension tubes. Retention of position was obtained by use of a posterior moulded plaster splint, extending from the middle of the upper arm to the distal ends of the metacarpals, with elbow flexed slightly less than 90 degrees, forearm being placed in supination.

Postoperative Course. This was uneventful, with no rise of temperature or other complication. Slight active motion in splint was started within twenty-four hours. This was increased as conditions permitted. The wound healed normally and the patient left the hospital on the tenth day. The splint was removed entirely at the end of the third week. Motion at this time was fair in all directions. Restriction in extension was the most marked impairment.

Three months after the injury all functions of the elbow were recovered, with the exception of complete extension, and this returned in an additional two weeks. One year after the injury, there was no impairment of any of the functions of the elbow joint.

CASE II. Mrs. H. M., aged 79 years, entered the hospital August 12, 1935, shortly after having fallen on her outstretched right hand. Examination showed gross deformity of both right wrist and elbow, with a small bleeding wound over the ulna about 2 inches below the olecranon. There was marked discoloration and swelling of the upper third of the forearm and about the elbow. No disturbance of the peripheral circulation or nerve function was noticed. The elbow was considerably widened with prominence of the internal condyle of the humerus. The forearm was pronated and definitely shortened. Diagnosis of external lateral dislocation of the elbow, with a Colles' fracture and a compounding wound of the ulna was made. X-ray study (Figs. 5–7) showed a complete external lateral dislocation of the elbow, with a fracture of the head of the radius. In addition, a Colles' fracture with marked displacement was noted.

Treatment. Under gas-oxygen anesthesia, the dislocation was easily reduced by using straight traction followed by flexion. The wound, which was not deep, was treated and tetanus antitoxin given. The Colles' fracture was manipulated into satisfactory position, and anterior and posterior moulded plaster splints were applied to the forearm. On account of the swelling about the elbow, the splints were not made to include that joint. The arm was placed in 90 degrees flexion. X-ray (Fig. 7) showed complete reduction of the dislocation, with satisfactory position of the bones involved in the Colles' fracture. The position of the head of the radius, while not entirely satisfactory, was considered to be the best that could be accomplished at the time.

The swelling receded rapidly and the wound caused no trouble. Guarded active motion was begun at the elbow on the second day and at the wrist on the third day. The patient left the hospital on the fifth day. Two months after the injury, there was good motion at the wrist with slight restriction in extension of the elbow. Flexion was complete. Supination of the forearm was somewhat restricted.

By February, 1936 there was complete restoration of function at the wrist and elbow except for restriction of supination. In August

plete external lateral dislocation of the elbow, complicated by avulsion of the epitrochlea.

Treatment. Several unsuccessful attempts

FIG. 20. Case VI. Lateral view showing marked comminution.

FIG. 21. Case VI. Anteroposterior view showing partial external lateral dislocation in subepicondylar position with a large fracture of external condyle and displacement of a smaller unidentified fragment below joint.

there was the same restriction of supination as at the previous examination. However, the function of the wrist and elbow was generally satisfactory, considering the injury encountered and the age of the patient. She was able to do light work about the house and was satisfied with the outcome.

Comment. This case is an example of the complete supra-epicondylar type and illustrates easy reduction, in spite of complicating factors, which adversely affected the outcome.

CASE III. M. S., aged 52 years, was admitted August 9, 1935, shortly after having fallen down a flight of five steps, landing on the left side with the entire weight of her body on the left forearm. Examination showed a deformity of the left wrist and marked swelling and widening of the elbow. There were also multiple lacerations about this region. No disturbance of peripheral circulation or nerve function was evident. The wrist had been fractured several years before, slight impairment resulting. X-ray examination (Figs. 8 and 9) showed a fracture of the distal end of the radius with position fair, and also an incom-

at reduction under the fluoroscope were made. Open operation was advised, but was delayed because of the condition of the skin about the elbow. On August 30, 1935, three weeks after the accident, open operation was performed. A posterior Kocher incision was used to expose the area. After cleaning out the olecranon fossa, reduction was easily obtained. No attempt was made to replace the epitrochlea. Position was maintained by the use of a posterior angular splint.

The postoperative course was uneventful, with no evidence of infection. Early motion of the wrist and elbow was started. On the patient's discharge from the hospital two and one-half weeks after operation, there was a fair degree of function, with, however, some limitation of flexion and extension.

Seven months after the injury, the patient was able to do her usual housework. Flexion of the elbow was complete, and extension was 75 per cent of normal. Pronation was complete with 75 per cent of normal supination. There was slight pain in the wrist. In August 1936, there was general improvement of all

functions, although there was still slight restriction of supination.

Comment. This case illustrates the type due

FIG. 22. Case vi. Lateral view after reduction showing considerable anterior displacement of external condyle. Later views indicated improvement.

to a fall directly on the elbow and forearm. We do not feel that the several attempts at reduction under the fluoroscope were wise, as the swelling and skin lacerations about the joint were aggravated, thus prolonging the preoperative period. Also we think it would have been more judicious to have dealt more definitely with the avulsed epitrochlea.

CASE IV. J. I., aged 14 years, was admitted October 28, 1935, shortly after sustaining a fall on his left elbow. Examination showed considerable discoloration, with widening and marked swelling of the elbow joint. The arm was held in moderate pronation. Peripheral nerve function and circulation were not disturbed. The diagnosis of external lateral dislocation of the elbow, with possible fracture of the olecranon, was made. X-ray study (Figs. 11 and 12) revealed a partial external lateral dislocation of the elbow, with a comminuted fracture of the external condyle, a fragment of the internal condyle lay in the olecranon fossa of the humerus.

Treatment. Under general anesthesia, apparently satisfactory reduction was obtained

after considerable difficulty. Excessive force was not used. X-ray (Fig. 13) showed reduction complete, with the fragment of the internal

FIG. 23. Case vi. Anteroposterior view after reduction.

condyle in good position. Because of the marked swelling, no attempt at splinting the elbow was made. The patient was put to bed with his forearm held at right angle flexion. Heat was applied and slight active motion was allowed on the second day. On the third day the swelling had subsided sufficiently to allow the application of a posterior moulded plaster splint, with a slight increase of flexion. The patient was discharged the same day, reporting to the Out-Patient Department two days later when increasing active motion was started.

At the end of the fourth week, motions of the joint were normal except for slight restriction of extension. Four and three-fourths months after the injury, examination showed complete return of function, with union of the replaced fragment of the internal condyle. (Fig. 15.)

Comment. This is an example of the type of injury due to a direct fall on the elbow. It indicates that, even in the presence of an interposed fragment, satisfactory closed reduction may at times be accomplished and that such a replaced fragment may become firmly reunited to the contiguous portion of the humerus.

CASE V. S. D., aged 56 years, was admitted September 30, 1935, several hours after a

fall on the outstretched left hand. There was deformity, with marked widening of the left elbow. A diagnosis of external lateral dislocation was made. No disturbance of the peripheral circulation or nerve distribution was found. X-ray (Figs. 16 and 17) showed a complete external lateral dislocation of the elbow, with a splinter fracture of the olecranon.

Treatment. Under general anesthesia, reduction was obtained without any difficulty, by using straight traction followed by flexion. X-ray check (Fig. 18) indicated complete reduction of the dislocation with the fragment of the olecranon in fair position. A posterior moulded plaster splint was applied with arm in acute flexion. Patient refused to be admitted to the hospital and was sent to her home some distance from the city. No further record was obtainable, so that the end results cannot be known. However, with the prompt and satisfactory reduction, we believe that a useful result was obtained.

This case represented the subepicondylar type.

CASE VI. Mrs. H. W., age 55 years, was first seen July 9, 1938, twenty-two hours after a fall on the right elbow and after an unsuccessful attempt at reduction. A description of the original deformity cannot be given. The original x-ray plate showed an external lateral dislocation, and a fracture of the external condyle with displacement of a large fragment laterally and a smaller unidentified fragment lying some distance below the joint. The unusual type of dislocation had not been recognized. At the time of examination there was no disturbance of peripheral circulation or nerve function.

Treatment. Under general anesthesia, strong traction with pronation followed by supination and flexion was made. Retake in operating room showed satisfactory reduction. Fixation in plaster splints, in acute flexion was applied. The patient was discharged the next day, with instructions as to early active motion. On August 16, function was good, with slight restriction of extension. By December recovery was complete.

Comment. This was a case of the subepicondylar type. In spite of delayed reduction, a good result was obtained.

SUMMARY

1. A study of external lateral dislocation of the elbow is presented, indicating that reported cases of both complete and incomplete dislocations are infrequent.

2. This type of dislocation is often complicated by fractures, in or about the elbow and at the lower levels of the forearm. Therefore, an x-ray study of the whole forearm should always be a part of the examination.

3. As early recognition is of importance, the chief of service should see such cases promptly.

4. Reduction, under general anesthesia, should be attempted at once, but the force exerted should be gentle.

5. If early closed reduction fails, open operation should be performed at once, especially if there is interposition of bone fragments.

6. Extensive skin lacerations and joint swelling, as a rule, contraindicate operation. However, operation may be considered in these cases if they are seen within three hours after accident, as tissues retain a resistance to infection within this period.

7. Open reduction permits inspection of the joint and removal of interposed tissues and extravasated blood, while reducing the possibilities of later joint changes.

8. Fixation should be maintained in supination with flexion to such an acute angle as may be permitted by the swelling.

9. It is a wise precaution to hospitalize these patients several days for observation.

10. Early active motion is essential in either open or closed reduction to obtain early restoration of joint function.

11. As in all elbow injuries, prognosis must be guarded.

12. Adequate follow-up supervision is indicated for a proper rating of end results.

13. Six case reports are given.

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I HAVE many times seen a doctor whose muscles are not 50 per cent as strong as the muscles of his patient, trying to reduce a fracture with his own hands without any form of mechanical contrivance to help him. From—"Fractures" by Paul B. Magnuson, third edition (Lippincott).

TREATMENT OF SHOULDER DISLOCATIONS

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IN the treatment of a dislocated shoulder, the surgeon must first consider his patient's safety and comfort in securing a good functional joint as rapidly as practical; and the surgeon must secondly protect himself against the ever-growing problem of malpractice suits. The patient is protected by care and attention to diagnosis, anesthesia, reduction, and after-treatment; the surgeon is protected by roentgenograms before and after reduction, and adequate records with full notes of any nerve or blood vessel injury, made before the arm is touched and preferably made before witnesses. Convenient methods are now available for printing data directly upon the emulsion of an x-ray film thus forming an unremovable label, difficult to falsify, upon which may be recorded the patient's name, date, hour, whether a pre- or post-reduction film, etc. The surgeon may thus further protect his x-ray films from being lost or mixed.

The following article presents a correlation of the author's experience, and some of the more recent methods of treatment described in the surgical literature. The presentation is from the viewpoint of the general surgeon or general practitioner who may lack the facilities and training of the orthopedic surgeon; special orthopedic skill is needed in the treatment of some of the more extensive shoulder injuries, but most shoulder dislocations can well be treated by the general surgeon. Closed reduction is advocated whenever practicable, because the end results of open reduction are not always gratifying, even in the best of hands. Closed reduction gives equal or better results with less immediate disability and less expense to the patient.

Etiology. Young adult males most commonly suffer humero scapular dislocations. Heavy labor or athletics often supply the needed violence. Muscular contractions of epilepsy or other seizures may also result in shoulder dislocations.

The classical mechanism resulting in this injury is a backward thrust on the hand or arm, when the arm is in a position of abduction and external rotation. The result is an anterior or subcoracoid dislocation of the shoulder; the humeral head is forced through the inferior aspect of the joint capsule and slips forward under the coracoid process.

The following case report illustrates that the muscle spasms of an epileptiform seizure may force the humeral head through the joint capsule, even in the absence of abduction.

CASE I. A 31 year old housewife complained of pain in her shoulder for five days, beginning immediately after she suffered a severe convulsive seizure in bed at night. The seizure was observed by the patient's husband who is certain that no abduction of the arm occurred. The patient had had previous epileptiform seizures but never any sort of shoulder injury. After recovery from the seizure, there was severe pain and limitation of motion of the left shoulder. Five days later, when she was first seen, a subcoracoid dislocation of the shoulder with avulsion of the greater tuberosity was found. Reduction was readily accomplished under brachial plexus anesthesia by the method of Zierold.[12]

Numerous other types of violence cause dislocated shoulders. Some unusual accidents and diseases causing this injury are: falls through a trap door or hole in the floor with arms forced above the head; puerperal convulsions; painting a ceiling; carrying a load on the shoulder; diving.

Diagnosis. The diagnosis of dislocated shoulder is usually easy to establish; mobility of the shoulder is very limited; there is a hollow beneath the acromion

process; the head of the humerus is palpable in its abnormal position, usually beneath the anterior axillary fold. The shoulder may be dislocated upward, downward, backward, or forward, but by far the most common is the forward or anterior dislocation.

In addition to the diagnosis of dislocation, any blood vessel, nerve, or bone injury must be determined. An anteroposterior roentgenogram will demonstrate avulsion of the greater tuberosity in about 7 per cent of cases,[10] and fracture of the surgical neck in about 0.5 per cent. Paresthesia or paralysis indicating brachial plexus injury occurs in 3 per cent. The radial pulse should always be felt, to help exclude pressure or injury of the axillary artery.

The value of roentgenograms both before and after reduction cannot be too strongly emphasized, both as an aid to accurate diagnosis and treatment and as a protection to the physician should his patient choose to allege that bony injury was caused or increased by the manipulation. The same is true of a careful record of evidence of soft tissue injury; the record should be made before any manipulation is carried out, and preferably in the presence of witnesses.

Treatment before Reduction. Reduction should be performed as soon as practicable; delay means prolonged disability. Pain should first be relieved by a hypodermic injection of morphine if it is necessary to transport the patient some distance for reduction or x-rays. It may be possible to reduce a dislocated shoulder with no other anesthesia in a fair proportion of cases, but as the suffering is usually severe, the author considers anesthesia advisable. Patients suffering from recurrent dislocation usually require no anesthesia, but in dislocations of previously well shoulders, much unnecessary pain can be avoided by the judicious use of some anesthetic agent, either regional or general.

Ethyl chloride used by inhalation to induce a state of analgesia has proved very

satisfactory in the author's hands in the reduction of recent dislocations. The equipment is extremely simple: a cylinder of ethyl chloride and a gauze mask. If the cylinder is placed in warm water for a few minutes before use, a freer flow of the drug is obtained. The patient's eyes are protected with a towel, and the mask is placed over his nose and mouth. The ethyl chloride is sprayed on the mask in a constantly moving stream to prevent freezing. The patient is instructed to count between breaths. When the counting stops or becomes confused, the patient is in an analgesic state much lighter than surgical anesthesia, and hence with a wider margin of safety. The dislocation may now be painlessly reduced, though the patient is often aware of what is going on and may even feel the "plop" that occurs when the head slips into place. The margin of safety of ethyl chloride is wide when used in this manner; the drug is contraindicated in the presence of liver damage or severe arterial disease; nausea and vomiting are uncommon.

For a more difficult reduction, such as one neglected for twenty-four hours or more, or one complicated by fracture of the surgical neck, a more prolonged anesthesia is needed. Ether or ethylene may be used. The author has found a regional nerve block permits deliberate manipulation without pain and avoids some of the risks and discomforts of general anesthesia; the method is particularly valuable when the patient has a contraindication to general anesthesia.

To induce brachial plexus block, the patient lies with his head drawn away from the involved side; a needle is introduced at a point 1 cm. above the midpoint of the clavicle; with the forefinger on the subclavian artery as a guide, the needle passes inward, downward, and backward in the direction of the fourth dorsal vertebra, until the first rib is reached. The rib usually lies at a depth of about 3 cm . If paresthesias of the arm occur, 15 or 20 c.c. of 2 per cent procaine hydrochloride solution with ad-

renalin are injected; if no paresthesias are obtained, 10 c.c. of the solution are injected, the needle reintroduced to strike the

FIG. 1. Hippocratic method of reducing shoulder dislocations.

lateral margin of the rib and another 20 c.c. injected here. This procedure differs from a block to anesthetize the arm; the branches of the branchial plexus to the shoulder joint come off high up and stream over the lateral margin of the first rib; a successful block of the shoulder joint may give incomplete anesthesia of the arm. Fifteen minutes should elapse from the time the procaine is injected until manipulation is attempted.

Bohler[5] injects 2 per cent procaine solution into the hematoma in fracture dislocations. Tietze[8] advises a wide infiltration of the shoulder muscles, as described below.

Reduction. Reduction may be accomplished either by traction, with or without pressure on the arm or the humeral head, or by manipulation to slide the head through the rent in the capsule. Kocher's maneuver is the most widely described method that uses manipulation without traction; briefly, it consists of external rotation of the arm which is flexed at the elbow, adduction of the elbow to the midline of the body, then

internal rotation of the arm. This method is very widely recommended and used, and its value is unquestionable. However, the
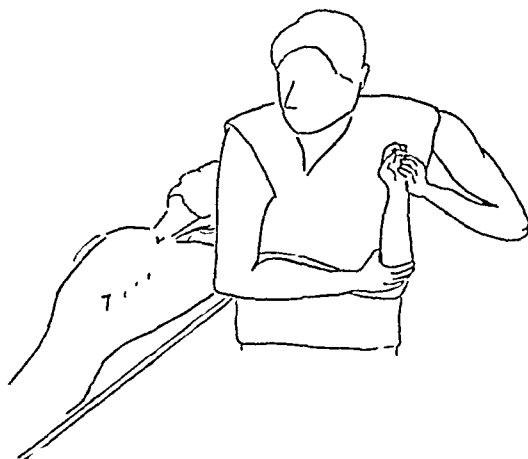
FIG. 2. Zierold's maneuver.

author has not found this maneuver universally successful. It may have to be repeated in order to effect reduction, and is not very useful in neglected dislocations. Because of the range of motion that the humeral head is put through during the manipulation, the possibility of considerable soft tissue damage exists, and there is some risk of fracture of the surgical neck. Kocher's method is contraindicated in dislocation with fracture of the surgical neck.

The Hippocratic method (Fig. 1) is a form of traction plus pressure on the humeral head. It consists of strong traction on the involved arm by the operator's hands which grasp the patient's wrist. Pressure on the humeral head is made by the operator's unbooted heel pressed into the patient's axilla. A slight levering motion of the arm toward the body helps to lift the head over the rim of the glenoid fossa. Bohler[5] advocates routine use of the Hippocratic method in recent uncomplicated dislocations. Though usually safe, the method carries some risk of brachial plexus injury by pressure of the heel in the axilla. The method is contraindicated in fracture of the surgical neck.

Traction at a right angle to the long axis of the body is free from the objections to Kocher's or the Hippocratic maneuver. Its

object is to overcome the resistance of the spastic shoulder muscles, free the head from where it is engaged, and allow the head to

months is so extensive as to make reduction exceedingly difficult. Conservative closed methods are indicated before operative
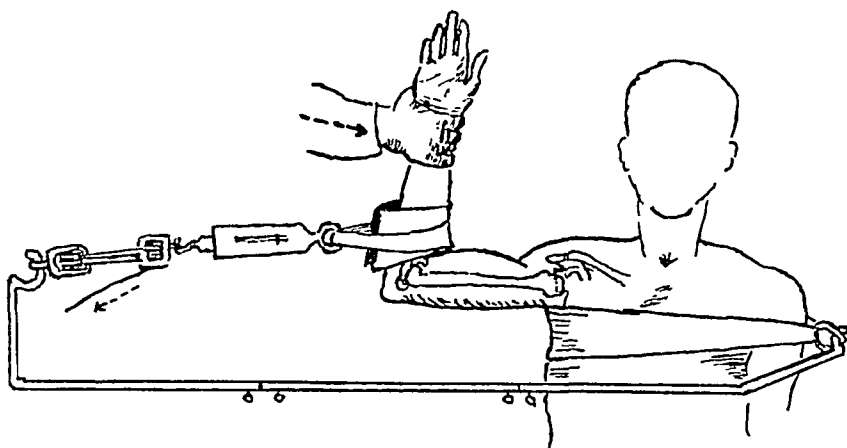
FIG. 3. Souter's traction apparatus, as applied to dislocations of the shoulder. Intermittent controlled traction is readily applied in the axis of the humerus.

fall into the glenoid. It is the only practical method of closed reduction of a fracture dislocation involving the humeral neck, and is quite satisfactory as a routine method in uncomplicated dislocations.

Lateral traction may be accomplished in a variety of ways, among which the most useful are: traction by the operator's hands upon the patient's wrist, with counter-traction by a swathe around the body; Zierold's[12] maneuver (Fig. 2); Souter's traction apparatus[2] (Fig. 3); screw traction with the leg supports of the Hawley table;[1] Bohler's traction frame.[5] Pressure on the humeral head may be used in combination with any of these forms of lateral traction, if traction alone fails.

Zierold's maneuver is particularly useful because it requires no apparatus other than the operator's hands and forearm and because it applies a powerful, easily controlled traction and countertraction. The operator's forearm (Fig. 2) forms a fulcrum for the patient's forearm which acts as a lever. It is effective in old dislocations if not too long neglected.

Treatment of Neglected Dislocations. The longer a shoulder remains dislocated, the more difficult becomes the reduction and the less satisfactory the end result. Fibrosis begins within ten days and within two

reduction should be considered unless it seems obvious that extensive changes have occurred that will prevent closed reduction.

Neglected dislocations require anesthesia, either regional or general. As soon as pain is controlled, the more simple methods of reduction should be tried. Zierold's maneuver is particularly valuable. Kocher's method or the Hippocratic method may be attempted but are much less useful than they are in recent dislocations.

Regele[7] of Bohler's clinic describes the following procedure, intended as an auxiliary method in the reduction of neglected dislocations: "After loosening the dislocation by traction and possibly by lateral displacement of the humeral head (with heel, or with both thumbs), the patient lies supine, the elbow of the flexed arm is placed so that its perpendicular projection falls approximately in the region of the sternoclavicular junction of the same side. The shoulder is firmly supported, and with strong, accurate pressure in the axis of the humerus, the head springs into place." (Fig. 4.)

Since excessive injury to the soft tissues is to be avoided, long continued or repeated manipulation is contraindicated. Lateral traction, either continuous or intermittent, will most often successfully reduce a

neglected shoulder dislocation, when more simple methods fail. Souter's traction apparatus, with 10 to 20 pounds pull, intermittently applied, is recommended by Thomas.[2] (Fig. 3.)

Bohler employs a screw traction frame with a transfixion pin through the olecranon process; this same frame can be left in place during an operative reduction, and facilitates freeing the head where it has become adherent and aids in moving the head into place. Screw traction may be obtained by fastening a swathe around the patient's body to one leg piece of a Hawley table and a hitch around his wrist to the other. The leg pieces are set at a right angle to the table and can be screwed apart, providing controlled traction.

A method for avoiding open reduction is described by Tietze and merits some consideration when one is faced with an exceedingly difficult reduction. He states: "—In our clinic we have established the following policy: the joint capsule is filled with 1/2 per cent novacaine without adrenalin, and the entire shoulder girdle musculature, particularly the subscapularis, is infiltrated with the same solution. Reduction is now attempted. Should this fail, we abandon the effort, and every two days, infiltration of the musculature is carried out. It then becomes possible to overcome the muscle spasm, rotate the head, and fix the arm in 60 degree abduction. After eight days, a second attempt at reduction is made, and in most cases the result is obtained."

Treatment after Reduction. The presence or absence of bone, blood vessel or nerve injury determines the plan of treatment after reduction. These complications are best treated on an abduction spint. In uncomplicated dislocations a simple sling suffices; no dressing is required after twenty-four hours. Active motion is then begun; the patient is taught certain simple exercises:

1. The patient leans forward, allowing his arms to hang loosely; he then fixes the position of his arms relative to his body,

and straightens his trunk. The result is that gravity has helped to move the arms to as much as a right angle with the body and
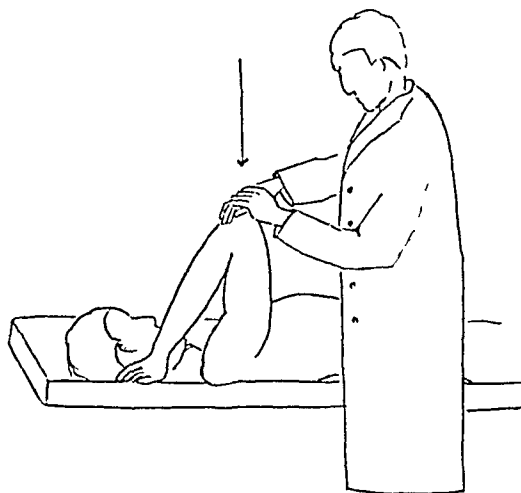
Fig. 4. Regele's maneuver for reduction of neglected dislocations. The elbow is placed in the vertical projection of the sternoclavicular joint of the same side, and a thrust made in the axis of the humerus; the head of the bone can thus be made to slip into the glenoid fossa.

the patient's muscles can then maintain the position although he is unable to move the arms against gravity to such a position.

2. The patient leans forward and swings a 2 pound weight, thus carrying the shoulder into abduction by gradually increasing the swing.

3. The patient stands with his injured shoulder towards a smooth wall; his fingers creep up the wall, thus abducting his shoulder; a mark is made at the highest point reached, and attempts are made each day to exceed the previous day's record.

4. With his arm at his side, the patient forces the back of his hand firmly against a wall—thus setting his deltoid without moving the shoulder joint. Deltoid setting may be begun the day of the injury to help avoid deltoid atrophy.

The patient's progress should be followed carefully, and should improvement fail to occur in three or four days, an abduction splint should be worn constantly except when exercising, until abduction to 140 degrees is possible. The arm should not be fixed at the side with a sling or a Velpeau bandage. In persons over 40, the abduction

splint is likely to be necessary Neurologic or vascular complications, as evinced by paresthesia, paralysis, or circulatory embarrassment, require an abduction splint until the symptoms of nerve or vessel damage disappear and until abduction is free.

No full discussion of complicating fractures will be undertaken here. The commonest bony injury is that of fracture of the greater tuberosity. Reduction is carried out under anesthesia, just as for uncomplicated dislocation. The arm is then put up in sufficient abduction to secure the reposition of the avulsed tuberosity. An axillary triangle is often sufficient; it may be conveniently combined with a Velpeau bandage made with 3 inch elastic bandage well pinned at all intersecting points. Occasionally, an abduction splint is necessary. Active motion of the elbow and wrist begins at once. The shoulder can be carefully moved in ten days, and exercises begun in three weeks. Union is solid in five weeks.

Fractures of the humeral neck, complicated by dislocation, are best treated by reduction of the dislocation with traction, and immobilization in traction on an abduction splint, the arm being rotated to secure accurate apposition of fragments. The elbow is moved at once; the shoulder is moved in three to four weeks, depending on the severity of the injury and the accuracy of reduction. The abduction splint is necessary for about five weeks.

Prognosis. The prognosis varies with the patient's age: persons under 50 years have less than one month of disability and should have no residual disability; those over 50 have two to three months disability, on the average, and may have a residual limitation of abduction.

The percentage of patients suffering recurrent dislocation is exceedingly difficult to determine and the author finds no published data regarding it.

Vascular complications, if properly treated, do not change the prognosis of a dislocation. Brachial plexus injuries may occasionally be permanent, but in most instances will recover fully if treated in abduction; for medicolegal reasons, a careful record of evidence of nerve injuries is essential.

Avulsion of the greater tuberosity means a period of disability of six to eight weeks; fracture of the surgical neck, twelve weeks. Young persons will retain full mobility of the shoulder. After the age of 40, there is likely to be some limitation of abduction.

Recurrent Dislocations. With present methods of treatment, some persons suffering one shoulder dislocation will experience the same injury repeatedly thereafter as the result of slight, often trivial, violence. Reduction in these cases is readily accomplished, but permanent relief is a more difficult problem.

A great variety of operations have been proposed to prevent recurrent dislocation, but all have been supplanted to a large extent by the Nicola operation. This procedure consists of exposing the shoulder joint, dividing the long head of the biceps, and transplanting its tendon from the bicipital groove into a canal drilled into the head of the humerus. The result is a. tendinous sling which prevents forward and downward motion of the humeral head, and dislocation cannot occur. The operation is notable for its successful end results with little or no limitation of motion as well as for its simplicity and safety. Epileptics are particularly subject to recurrent dislocations and can well be treated by the Nicola operation.

SUMMARY

1. Treatment of a dislocated shoulder involves diagnosis, anesthesia, reduction, and after-care.

2. The surgeon must protect himself with x-ray and written records of bone, nerve, and blood vessel injuries.

3. Brachial plexus anesthesia is a convenient, efficient, and safe method for controlling the pain of shoulder joint reductions.

4. Traction, either by Zierold's maneuver or by mechanical devices, is efficient in effecting reduction.

5. After treatment is important in obtaining good results; early active motion and exercises are advised.

6. Prognosis is better in younger individuals and in recent dislocations.

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ANALYSIS OF RESULTS FOLLOWING SYMPATHECTOMY FOR PERIPHERAL VASCULAR DISEASE*

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MATERIAL STUDIED

THIS report is based on a follow-up study of 105 sympathectomies done on fifty patients suffering from various types of peripheral circulatory disturbances.

TABLE I

INDICATIONS OF SYMPATHECTOMY FOR PERIPHERAL VASCULAR DISEASE
1928–1936

Diagnosis	Selection of Cases
Raynaud's phenomena	Lack of marked structural changes
	Absence of sclerodactylia
	Stages 1 and 2
Buerger's disease	Absence of acute inflammatory stage
	Below 40 years of age with definite collateral reserve
	Poor response to conservative treatment
Poliomyelitis	Moderate paralysis limited to one extremity; evidence of vasospastic phenomena
	Age preferably between 6 and 10 years
Reflex dystrophy (causalgia, traumatic vessel spasm, Sudeck's atrophy).	Severe cases producing disability, resistant to physiotherapy, exhibiting abnormal vasomotor phenomena
Unclassified . .	Rapid onset of digital thrombosis with impending gangrene
	Mostly upper extremities involved

In the group diagnosed as Raynaud's disease (Table 1), a number of patients exhibiting Raynaud's phenomena due to secondary vessel spasm were first excluded. The diagnosis of Raynaud's disease was only arrived at by exclusion. In the typical forms, the stage of sclerodactylia with deep ulcerations was not subjected to operation, as our results in this group have been unsatisfactory. Operation for Raynaud's disease seemed most successful in the patient in whom organic obstruction of the digital arteries was absent or slight and in whom involvement of the skin, joints, and tendons had not yet taken place. Between these two groups there is a third, in which there is some organic damage, but dilatation of the vessels by reflex heat or vasodilators was still possible but incomplete. This group shows such improvement that operations are justified.

Operations on patients suffering from Buerger's disease have not been advocated in: (1) an acute inflammatory stage; (2) in a late stage with a closed collateral bed (frozen vascular tree); (3) patients past the age of 40; (4) if economic status permits a long rest in an even warm climate or if the cardiovascular apparatus is diffusely involved.

It is most difficult to argue for or against a sympathectomy in patients around 40 years of age who have had an acute arteritis in the early twenties, whose disease has been quiescent for many years and now present themselves with a second flare-up, usually in another previously uninvolved extremity. Such patients show little capacity for vasodilatation and show some sclerotic changes. They may have myocardial damage. In such cases, intensive conservative treatment together with paravertebral alcohol injection has arrested the disease in fifteen patients. This method should certainly be tried before sympathectomy in such borderline cases.†

† Paravertebral injections of alcohol as advocated by Reichert[1] have been done on twenty-five patients suffering from obliterating arteriosclerosis and are discussed elsewhere.[2]

We have urged sympathectomy, however, in individuals between 20 and 40, who are having their first attack and who have a good capacity for vasodilatation. The result of the sympathectomy mainly depends on the stage of the disease, which may differ in each of the four extremities.

In case the upper extremity is involved, which is more frequent than suspected, sympathectomy is done as soon as nutritional changes, such as atrophy, ulceration or gangrene are manifest.

Following operation, the same conservative régime is instituted as preoperatively and is followed for many years.[3] This consists of complete abstinence from tobacco, increased fluid and salt intake, a series of typhoid vaccine injections twice a year and intermittent venous hyperemia treatments to stretch the venocapillary bed. This last method is more effective in the sympathectomized patient, where the resistance to stretch has been diminished.[4]

In poliomyelitic children, if they are younger than 8 years, if the paralysis is limited to one extremity and is moderate in degree, and if their extremities are cold, plum-colored and painful, if nodular patches of cyanosis, edema and chilblains are present, a sympathectomy will provide increased vascularity. An acceleration of growth, as reported by Harris[5] has not been observed in our cases.

In the fourth group, I have gathered a group of vasomotor and nutritional distubances, diagnosed as Sudeck's atrophy, traumatic osteoporosis, stump-neuroma, and causalgia and have called them reflex dystrophies.[6] Not only does the edema, cyanosis, and sensitivity to heat disappear, but bone may recalcify, as shown by Fontaine and Herrmann.[7]

A group of "unclassified" cases had to be established in this material. Some of the most dramatic successes have been obtained in this group without, however, establishing the possibility of a definite preoperative diagnosis.

ANALYSIS OF RESULTS

It is quite difficult to evaluate any method used in the treatment of peripheral vascular disease. Silbert[8] has recently pointed out that unless certain factors are eliminated, the value of any treatment must remain dubious. These factors are (1) the tendency to spontaneous improvement; (2) the effect of cessation of smoking; (3) the normal variations in vasoconstriction due to environmental changes in temperature and the patient's psychic state. To this one may add the effect of (1) rest in bed, (2) mild heat, (3) plenty of fluids, (4) regulation of the patient's diet, and (5) the surgical care of ulceration and necrosis. Obviously a complete elimination of all these factors in analyzing one form of therapy is almost impossible.

In trying to find out the place of sympathectomy in the treatment of peripheral vascular disease, further difficulties present themselves. A good result in Raynaud's disease means freedom from painful vasospastic attacks and softening of the sclerotic digits; but in patients suffering from Buerger's disease an arrest of the disease and the avoidance of amputation must be regarded as a good result even if a functional restoration to normalcy is an anatomic impossibility. That proper selection of cases, technically complete operations, avoidance of regeneration and adequate postoperative follow-up are important factors in obtaining good results has been pointed out elsewhere.[9]

In spite of the multiplicity of variables, an attempt has been made to evaluate the results of sympathectomy. It has been done by utilizing several methods. *First,* objective evidence has been sought that peripheral circulation has been modified by the operation. *Second,* in patients suffering from symmetrical lesions of approximately identical severity, one extremity was left for control over a period of several years. *Third,* the total material of 105 sympathectomies performed on fifty patients suffering from various types of peripheral vascular

disease has been followed for at least two years and the results tabulated according to criteria to be discussed.
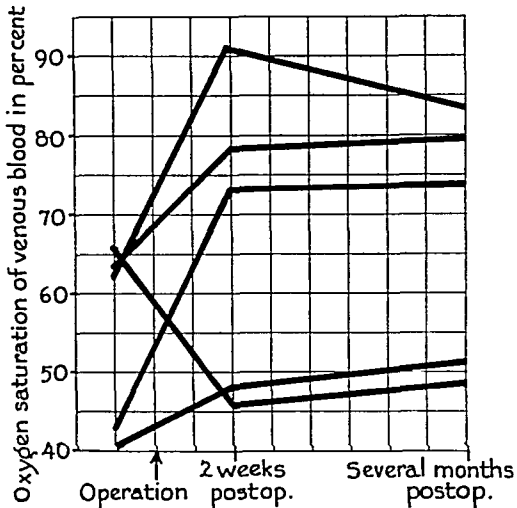
FIG. 1. O_2 saturation of venous blood taken from the femoral vein before, two weeks and several months after sympathectomy. Note the marked rise in the first three instances, a moderate rise in the fourth and a definite drop in the fifth case. This last patient developed a popliteal thrombosis following operation which, however, did not result in gangrene.

THE MODIFICATIONS OF CIRCULATION FOLLOWING SYMPATHECTOMY

The blood vessels of the sympathec-tomized limb react to direct application of heat and cold and also to ischemia in the normal manner.[9] But, being deprived of their vasomotor innervation, these vessels are freed of a number of extrinsic and intrinsic stimuli, which arrive to the vessels over the efferent sympathetic path-ways. Thus cooling or pinching the body,[10] pain, fright or anger[11] do not produce vasoconstriction in the sympathectomized extremity, nor will heating the body[12] or the production of fever with typhoid vaccine[13] produce vasodilatation. The ques-tion arises, how important are these factors in regulating circulation? When studies in blood flow are made with a modified Hewlett-van Zwaluvenburg method, it becomes apparent that great fluctuations in blood flow are produced by the stimuli enumerated above. Sympathectomy abol-ishes these fluctuations. In a recent study of

some of my patients, Gellhorn and Steck[19] found that the inhalation of CO_2, which normally produces a marked central vaso-constriction of peripheral vessels, is abol-ished in the completely sympathectomized extremity and may even give rise to a paradoxical vasodilatation as the local vasodilating action of CO_2 may now become manifest. This method may serve as an index for the completeness of sympathetic denervation.

Another effect of a permanent vasomotor palsy can be detected by the study of blood gases, notably the oxygen saturation of the venous blood (Fig. 1) draining the sympathectomized extremity. Detailed fig-ures have been presented elsewhere.[9] A simple graph illustrates some data ob-tained on four patients by Dr. F. K. Hick, all of whom had arterial and venous (fe-moral) punctures before, shortly after and several months after the operation. The degree of the rise depends on available collaterals and corresponds closely with the clinical improvement. In one case shown here, the oxygen saturation dropped from 66 per cent to 47 per cent. An explana-tion of this was found in an acute popliteal thrombosis following the operation, which, however, did not result in gangrene.

THE CLINICAL COURSE OF THE DISEASE IN UNILATERAL SYMPATHECTOMIES

Sympathectomy according to our present knowledge does not remove diseased struc-tures. While some authors have reported inflammatory or degenerative changes in the ganglia removed from Raynaud's disease or Buerger's disease, other studies, notably those of Craig and Kernohan,[14] revealed no pathologic changes. Our mate-rial has been studied with neurohistologic methods by Dr. Hassin, who has never reported any abnormal histologic findings. The vasomotor palsy which is obtained by sympathectomy has a favorable effect on the circulation of an affected limb even though it does not strike at the cause of the disease. There are eight patients in this series, who either purposely or uninten-

tionally have one denervated extremity, while the other one has been left alone or sympathectomy has been incomplete. The

Six of these patients suffer from Raynaud's disease. The operated side is completely dry, warm and the texture of the
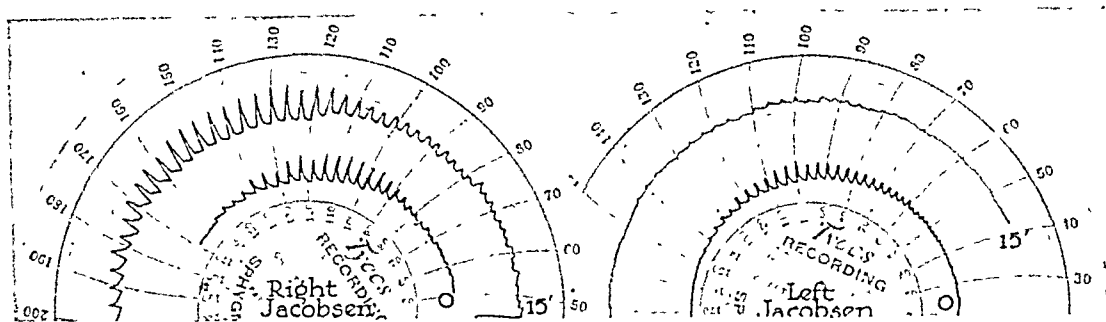
FIG. 2. Effect of reflex cold on the sympathectomized and control extremity. Right lower extremity sympathectomized several months previously. Note marked vasoconstriction in the control extremity in contrast with paradox increase in pulsation on right side. This increase is due to the systemic rise in blood pressure which followed the immersion of both hands in ice water.

FIG. 3. Failures of sympathectomy affected by preoperative diagnosis. Note that Raynaud's disease shows a failure of 15 per cent which is partly due to incomplete operations and partly to the fact that most of these were cervicodorsal sympathectomies which give notoriously poor results. The unclassified group shows the largest percentage of failures because most of them are probably not of vasospastic origin. (See also Table IV.)
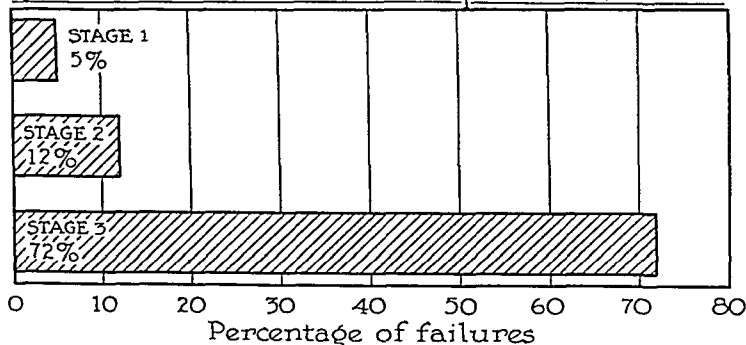
FIG. 4. Table III in graphic form.

operated side has usually been more involved by the disease process than its fellow.

skin is looser. Color changes may still occur, chiefly at the tips of the digits, but are not painful. The burning on release of

the spasm is absent. The control side sweats profusely, spasms occur on emotional stimuli or environmental changes;

standstill. In another patient, who had lost both feet before coming under observation, one upper extremity has been sympathec-
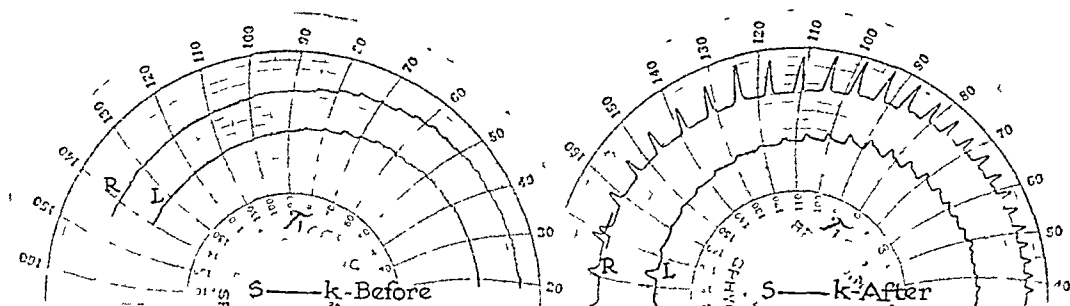
FIG. 5. Oscillometric curves of a 21 year old patient suffering from Buerger's disease. Note the marked opening of the vascular bed fifteen minutes after the intravenous injection of sodium nitrite on the right side. The left side hardly responded to the vasodilator. This patient's right foot is in an earlier stage of disease than the left; better results may be expected from sympathectomy on this side.
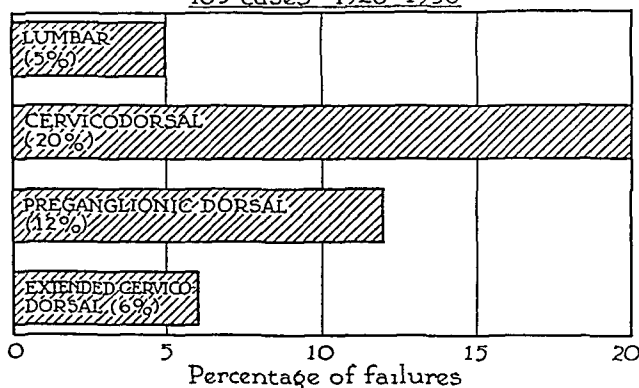
FIG. 6. Illustrating the percentage of failures (Table IV) in graphic form.

the sclerodactylia slowly progresses; small ulcers heal slowly or not at all. The same picture is true of the incompletely sympathectomized extremity. Patients who are well selected invariably request operation on the control side at an early date.

Two patients suffering from Buerger's disease have had unilateral lumbar sympathectomies. In both instances the side which was far more affected and had shown ulceration was operated upon. In one patient who has now been followed for three years and has been getting all the benefits of conservative treatment, the sympathectomized limb is now warmer, the ulcer is healed and his walking ability could be improved by intermittent venous hyperemia. The other extremity is at a

tomized; in that hand there were multiple ulcers and continuous pain, both of which were favorably influenced. The control side, in spite of all conservative treatment is slowly progressing. These patients are especially mentioned because the argument that conservative measures used in conjunction with sympathectomy might have been the cause of improvement cannot hold here.

As pointed out before, sympathectomy inhibits the fluctuations of vasomotor tonus; these fluctuations are intensive enough to elicit a mechanism which sympathectomy abolishes in Raynaud's disease. In Buerger's disease (Fig. 2), however, there is only infrequent evidence of acute vasospastic phenomena; there seems to be

a more chronic continuous sympathetic stimulation originating reflexly from segmental thrombi. Such a reflex vasoconstriction can be interrupted by local excision of thrombosed vessels, by a spinal anesthetic or by a sympathectomy.[6] The last, of course, is the most feasible and permanent.

ANALYSIS OF THE WHOLE MATERIAL

Only patients operated on before the end of 1936 have been included. (Table II.)

TABLE II

RESULTS OF SYMPATHECTOMY FOR PERIPHERAL CIRCULATORY DISTURBANCES

1928–1936

Diagnosis	No. of Patients	No. of Operations	Success	Improvement*	Failure	Mortality
Raynaud's disease.	10	26	17	4	5	
Buerger's disease..	21	56	49	4	2	1
Poliomyelitis with vessel spasm....	3	3	3			
Reflex dystrophy (causalgia, osteoporosis)	10	10	10			
Unclassified	6	10	7	1	2	
Total	50	105	86	9	9	1

* Improvement indicates definite amelioration of symptoms but not complete relief. Failure indicates that the disease had either progressed or remained at its preoperative state. The only death occurred five days after lumbar sympathectomy, due to coronary thrombosis.

Of the many variables, I have picked three factors for analysis, and the results have been tabulated accordingly.

The *preoperative diagnosis* modifies the obtained results in that there is the largest percentage of failures in the unclassified group, still a considerable percentage in Raynaud's disease and very encouraging results in Buerger's disease. (Fig. 3.) This rather startling result is explained first of all by the fact that most operations in the unclassified group and in Raynaud's disease were on the upper extremity which is notoriously more difficult to denervate, and secondly by the very rigid criteria which are used to determine the operability of Buerger's disease. Also the unclassified group will gradually be eliminated by a definite etiologic diagnosis and will form a part of the non-operative group in the future.

The *stage of the disease* in which the operation is undertaken (Table III) and

TABLE III

FAILURES OF SYMPATHECTOMY INFLUENCED BY DEGREE OF STRUCTURAL INVOLVEMENT

(105 Operations 1928–1936)

Stage	Criteria	No. of Operations	Failures, Per Cent
1	No structural disease Vessels dilate fully	22	5
2	Moderate structural changes Incomplete capacity for vasodilation	76	12
3	Advanced structural changes* Minimal capacity for vasodilatation	7	72

* Note that in Group 3 the percentage of failures is 72. Obviously operations in this group are justified only in an attempt to save the leg from amputation. Note also that most patients belong to the second group.

the spontaneous course of the disease at the time and following the operation are important factors in the end results. (Fig. 4.)

TABLE IV

TYPE OF OPERATION AFFECTING RESULTS

Operation	No. of Operations	Failures*
Lumbar sympathectomy.....	40	2 (5 per cent)
Cervicodorsal sympathectomy	25	5 (20 per cent)
Preganglionic dorsal sympathectomy..............	24	3 (12 per cent)
Extended cervicodorsal sympathectomy..............	16	1 (6 per cent)
Total...................	105	11 (10 per cent)

* Failure of sympathectomy: no change from preoperative status or progress of deterioration. Note that lumbar sympathectomy and the extended cervicodorsal sympathectomy have the lowest percentage of failures.

Patterson Ross[15] has classified Raynaud's disease into three groups and I believe that this could be done for all peripheral circulatory disturbances. According to the predominance of the vasospastic against the structural element, the success of sympathectomy in the different groups is striking. In the first stage there is no or

very little detectable change in the arteries or in the soft tissues of the digits. (Fig. 5.) The vessels dilate fully when heat is

FIG. 7. Diagram of cervicodorsal sympathetic chain. 1, stellate ganglion. 1a, intermediate ganglion, 2, 3, and 4, second, third and fourth dorsal ganglia. A, cervicodorsal sympathectomy. B, preganglionic sympathectomy. C, extended cervicodorsal sympathectomy. This last operation has given us results in sixteen cases which are comparable to those obtained by the lumbar sympathectomy.

applied reflexly, when the sympathetic fibers are blocked with novocaine or when sodium nitrite is injected intravenously. In the second stage there is ulceration of the tips of digits or painful stellate scars. The vessels show capacity for vasodilatation but it comes on slowly and is incomplete. In the third stage patients have advanced structural disease of the arteries. The vessels only have a minimal or no capacity to dilate. The soft tissues are atrophic or sclerotic and the tips of the digits are ulcerated or gangrenous.

Most patients both in private and dispensary practice belong to the second and third group. The first group may not see a surgeon nor is it easy to determine that arrest of the disease might not take place. In this group conservative measures carried out for a sufficiently long time, give one an insight into the spontaneous course of the disease. Obviously operations in the third

group are only justified in an attempt to save the leg from amputation. Unless a surgeon is thoroughly familiar with the
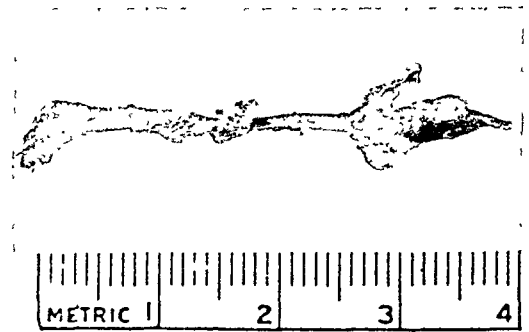
FIG. 8. Extended cervicodorsal sympathectomy performed through the anterior, supraclavicular approach. The chain was cut above the stellate and below the third dorsal ganglion.

course of peripheral vascular disease, its remissions and exacerbations, he will operate either too often or not often enough.

The type of operation performed is the third and last factor (Table IV) we have analyzed. Lumbar sympathectomy, in the consensus of all workers (Fig. 6), has always given superior results to sympathectomies done for the upper extremities. As shown in the graph, lumbar sympathectomies, most of which were done for Buerger's disease, poliomyelitis or causalgic syndromes, have been quite successful. The cervicodorsal sympathectomies have been done in three different ways. (Fig. 7.) The diagram to the left illustrates the removal of the stellate ganglion; the dorsal chain is sectioned below the second thoracic ganglion. This is Adson's method except that a cervical approach is used in my clinic. The diagram in the middle shows the principle of the preganglionic sympathectomy as advocated by Telford. The sympathetic chain is cut below the third ganglion, and the third and second white rami are cut. This would leave the postganglionic fibers intact. It is stated by J. C. White[16] that such postganglionic degeneration which occurs after a typical cervicodorsal sympathectomy is the cause of recurrent spasms in Raynaud's disease as the vessels are now sensitized to epine-

phrine. The diagram to the right shows my present method in which the excision is extended upward to include the inter-

will rise enormously after sympathectomy from 50 to 70,000 ohms to 300,000 ohms or more. (Fig. 9.) For practical purposes,
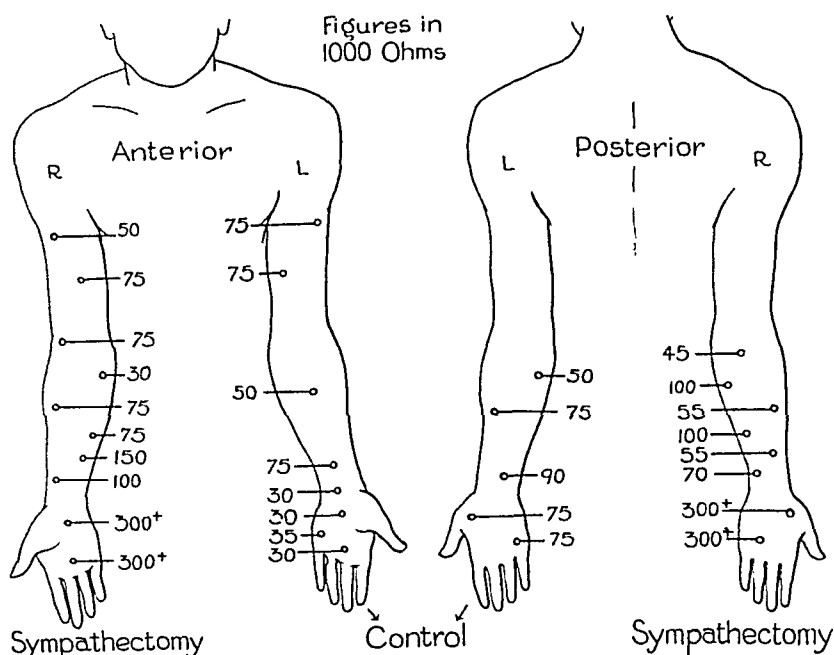
Fig. 9. Determinations of galvanic skin resistance on the control and on the sympathectomized side. Note that on the palmar surface of the operated hand the skin resistance rises above 300,000 ohms. Note also that on the ulnar surface the readings are equally high, indicating a complete operation. The skin resistance on the lower and upper arm did not seem to rise at all.

mediate ganglion or if this is invisible to strip the vertebral artery and caudalward it includes the third thoracic ganglion. (Fig. 8.) My results with this last type have been better than with the preganglionic method, which does not cut the important first white ramus,[17] so that some sweating remains in the hand. Since the recurrence in Raynaud's diseases following operation occurs within six months or a year and sensitization phenomena appear six to eight days after operation,[18] the sensitization cannot be considered a sign of recurrence in the postganglionic sections.

The completeness of the operation either on the lower or on the upper extremity can be demonstrated by Minor's starch-iodine or Grace Roth's cobaltous chloride test. A very accurate and numerical expression of the completeness of sympathectomy can be obtained by determining the resistance of the skin to the galvanic current. This

however, the palpating finger can readily detect a strip of moisture on the sympathectomized limb. On the upper extremity this moist strip is usually of ulnar distribution, on the lower it follows the femoral or saphenous nerve. This has led us to take special care in removing all of the second and preferably the third thoracic ganglion on the upper extremity and the second or even first lumbar ganglion for the lower extremity.

This technical discussion is important because of my conviction that failures, at least in this material frequently occur in insufficiently sympathectomized limbs. A completely dry hand has not ever become moist—the longest observation being eight years. But a slight area of perspiration left on a sympathectomized limb will grow as if regeneration could occur from an overlooked fiber. In evaluating results this point requires consideration.

SUMMARY AND CONCLUSIONS

As a result of this study it is felt that sympathectomy has a definite place in the treatment of peripheral circulatory disturbances. The results are influenced by the type and extent of the disease, by the natural course of the disease, by the extent of the operation and some other factors which elude statistical analysis and have to be determined in the individual case. Sympathectomy should be undertaken by men who are fully familiar with peripheral vascular syndromes, their natural course and their response to other forms of therapy. Undertaken under such conditions sympathectomy shows an increasing percentage of successful results.

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THE SURGICAL TREATMENT OF CARCINOMA OF THE PANCREAS

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WITH the technical developments which in the past few years have made possible radical resection of carcinomas of the pancreas, with the discovery of vitamin κ and its clinical application in the control of the bleeding tendency in obstructive jaundice, and finally with improved preoperative and postoperative management of jaundice, there has been an increased interest in the surgical treatment of carcinoma of the pancreas. Operations which five years ago would have entailed too much risk to justify their performance can now be carried out with relative safety. What is now required is a means for the recognition of carcinoma of the pancreas in the stage where it is still amenable to surgical excision.

Carcinoma of the pancreas does not differ in any essential way from carcinoma in other organs. It begins as a localized lesion and in a reasonably high percentage of cases it is a slow-growing, well-differentiated tumor. Metastases are present in a large number of the cases, mainly because the carcinoma gives few symptoms in its early stages and is therefore recognized late in its clinical course. Like carcinoma of the thyroid gland, no alarming symptoms are observed by the patient until the disease has progressed to such an extent that vital structures are invaded.

Again, like carcinoma of the thyroid, carcinoma of the pancreas gives definite premonitory symptoms which, if properly interpreted, will lead to earlier recognition and more effective treatment. Relatively early in the course of the disease, patients with carcinoma of the pancreas often experience symptoms of sufficient severity to bring them to the physician and it is the physician who may be at fault for not interpreting these symptoms correctly. In the past the physician has been loath to make a diagnosis of carcinoma of the pancreas because of the uniformly hopeless prognosis in this disease. Recognition of carcinoma of the pancreas and confirmation of the diagnosis by exploratory operation have been equivalent to the signing of the death warrant. But now that early recognition of this lesion offers the possibility of a surgical cure, it is essential that we utilize every means at our disposal to confirm the diagnosis.

DIAGNOSIS

In approximately 30 per cent of the cases of carcinoma of the pancreas in the Cleveland Clinic series, jaundice has not been present at the time of the patient's death or at the time of confirmation of the diagnosis by exploratory operation. In many of these cases the disease apparently originated in the body or in the tail of the pancreas. Pain was the most common symptom in this group of cases, was usually referred to the epigastrium, and was of a constant, deep, boring, quality; it was unrelated to meals and was present at night as well as in the daytime. In a smaller group of cases metastasis to distant points (brain, vertebrae, sternum, etc.) was the leading finding.

The prognosis in these cases of carcinoma of the pancreas without jaundice in which the tumor originates in the body of the organ or in such a location that the common bile duct is not involved, is nearly certain to be unfavorable. The disease does not manifest itself until extension or metastases involve vital structures at which time the possibility of cure is

past. It is in the highly differentiated, slowly growing tumors of the head of the pancreas that the common bile duct becomes obstructed and the diagnosis can be established before the disease has progressed beyond the stage of curability.

Although the development of jaundice has long been considered to be the cardinal sign of carcinoma of the pancreas, an analysis of any group of cases will show that, in a high percentage, symptoms are present for some time prior to the onset of the jaundice. These "prodromal" symptoms consist of anorexia, weakness, fatigue, loss of weight, and vague distress in the epigastrium; these were present in 72 per cent of the cases in this series and antedated the jaundice by from one to ten months. These symptoms were usually of sufficient severity to bring the patient to a physician, but in only a small percentage of the cases was the correct diagnosis suspected until jaundice appeared. It is in this group of cases that the diagnosis often can and should be established earlier.

If a man beyond the mid-period of life suddenly and unaccountably begins to fail in weight and strength and if marked anorexia and vague epigastric distress are present, carcinoma of the pancreas should be suspected. If careful clinical, Roentgen, and laboratory studies fail to reveal any cause for the patient's symptoms, if there is no psychic background for the complaints, and if rest and diet do not result in a prompt and striking improvement, an exploratory operation to determine whether early carcinoma of the pancreas is present should be advised.

The development of these "prodromal" symptoms of carcinoma of the pancreas coincides, in all probability, with the occlusion of the pancreatic duct by the tumor. Since the pancreatic duct is occluded within the substance of the gland, the disease is still potentially curable by resection of the head of the pancreas. It is at this stage of the disease, before the onset of jaundice, that the best surgical results will be obtained.

Although severe pain occurred in only 36 per cent of the cases of carcinoma of the pancreas in this series, a vague distress in the epigastrium, usually coming on at the onset of the "prodromal" period, was a common complaint. Intermittent occlusion of the pancreatic duct no doubt gives rise to painful sensations and to reflex disturbances of the gastrointestinal tract just as an intermittent hydronephrosis may cause pain and gastrointestinal symptoms. When the occlusion has become complete, the pain as a rule subsides and does not recur until the carcinoma invades the surrounding structures.

In addition to the presence or absence of pain, several points are of value in differentiating carcinoma of the pancreas from a stone in the common duct. The age of the patient is not of much value, the average age of patients with stones in the common duct being 52 years as compared with 56 for carcinoma of the pancreas. In this series a higher percentage of patients under 40 years of age had carcinoma of the pancreas than choledocholithiasis.

Carcinoma of the pancreas is predominantly a disease of men; only 16 per cent of the cases in this series occurred in women.

The depth of the jaundice and the continuity of the jaundice after its initial appearance are important factors in differentiating the two conditions. In no case of carcinoma of the pancreas did the jaundice disappear after it was once established, whereas intermittent jaundice accompanied often by chills and fever was a common finding in the presence of choledocholithiasis. Fever was rarely associated with carcinoma of the pancreas, and when present was indicative of extensive metastasis.

In the patients with carcinoma of the pancreas, the jaundice was usually very deep as a result of complete obstruction of the common bile duct. In this group of cases the icterus index averaged 100 units

as compared to 45 for the cases with calculi in the common duct. Unless the patient is seen during the first few days of the jaundice, icterus indices of less than 50 are rare in carcinoma of the pancreas. Pruritus tends to be more marked in carcinoma of the pancreas and may occur before the patient notices the jaundice.

In 90 per cent of the cases of carcinoma of the pancreas having jaundice, the liver is palpably enlarged, usually as a result of biliary obstruction rather than metastasis and this enlargement is more marked than is usually found in the incomplete obstructions associated with choledocholithiasis. Only 27 per cent of the patients with common duct stones had a palpable enlargement of the liver.

Courvoisier's law, although often inaccurate before operation as a result of difficulties experienced in palpating the gall-bladder through a thick abdominal wall, holds surprisingly true when judged by the operative findings. Gallstones will be found in approximately 20 per cent of the cases of carcinoma of the pancreas and in about half these cases the gall-bladder will be contracted as a result of inflammation from the stones. In another small group of cases, previous operations (cholecystostomies, etc.) will have resulted in contraction of the gall-bladder. In a third small group of cases, the carcinoma will be so extensive that the cystic duct as well as the common duct will be occluded by the tumor, and under these circumstances the gall-bladder will not be dilated. With the exception of these three groups, Courvoisier's law was found to hold true in all cases in this series, the gall-bladder being dilated in 69 per cent of the cases of carcinoma of the pancreas. The gall-bladder was dilated in only one case of stone of the common duct and in this case the head of the pancreas was extremely hard and the presence of a carcinoma could not be ruled out.

In the presence of complete obstruction of the common bile duct, duodenal drainage gives no information except the negative fact that no bile is coming through—a point indicating the presence of malignancy. If cholesterol crystals are recovered in the duodenal bile the presence of a stone in the common duct is suggested.

PREOPERATIVE MANAGEMENT

An analysis of the causes of death following operations performed on patients with obstructive jaundice shows that the most dangerous complication is postoperative hemorrhage, this complication alone having been a factor in one-third of the postoperative deaths. Until recently we have had no reliable method of determining the presence of a hemorrhagic tendency and no effective method of correcting it. Routine laboratory methods for the determination of bleeding and clotting time have been of no value. The Lee White method, dependent on the coagulation time of venous blood, has been of limited value. The only reliable method of recognizing a hemorrhagic tendency is by the prothrombin clotting time of Quick.[1] This subject has been discussed in detail in a previous publication.[2]

The bleeding tendency in jaundice is the result of a deficiency of prothrombin. Normally this substance is present in the plasma in approximately five times the quantity necessary to insure normal coagulation. Little or no change in the coagulation of the blood is noted until the prothrombin falls to 20 per cent of its normal level, but when it falls below this point a marked hemorrhagic tendency appears. This hemorrhagic tendency can be diagnosed with accuracy by the prothrombin clotting time.[1]

It has been well established by both animal and clinical investigations that the lowered prothrombin levels and the hemorrhagic tendency in obstructive jaundice are the result of a failure in the absorption of vitamin K from the intestinal tract. This substance is a fat soluble vitamin and is not absorbed unless bile is entering the intestinal tract. Butt and Snell[3] have clearly shown that, when

vitamin K and bile salts are given to patients with obstructive jaundice, the hemorrhagic tendency is corrected and the
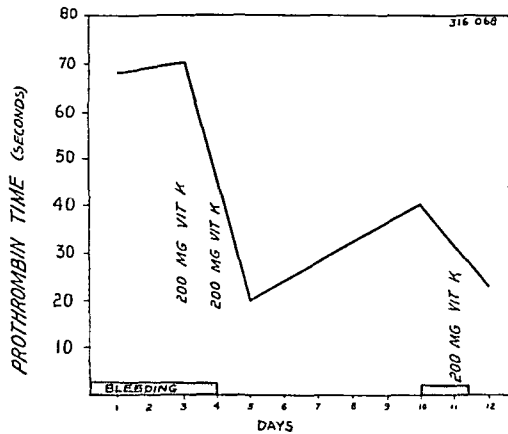
FIG. 1. Chart showing the reduction in prothrombin clotting time and cessation of bleeding following the administration of vitamin K.

prothrombin clotting time falls to normal levels. Figure 1 indicates the response of the prothrombin clotting time of a jaundiced patient to the administration of vitamin K and bile salts. Prior to the administration of vitamin K the patient had been bleeding from the incision, from the gastrointestinal tract, from the uterus, and from all mucous membranes. Within twelve hours after the oral administration of 200 mg. of vitamin K all bleeding stopped and the patient went on to recovery. As Butt and Snell have emphasized, it is essential that the bile salts be given with the vitamin K in order to insure its absorption.

In view of the importance of hemorrhage as a postoperative complication in patients with obstructive jaundice and in view of the effectiveness of vitamin K in the prophylaxis and treatment of hemorrhage, it is clear that operation should never be performed on jaundiced patients unless the prothrombin clotting time is normal or has fallen to normal levels in response to treatment with vitamin K.

The second consideration in the preoperative preparation of the jaundiced patient is the status of the liver. In order to insure the best possible liver function, the diet should be high in carbohydrates

and in total calories. In addition a 10 per cent solution of glucose should be given intravenously each day for several days until the glycogen reserve of the liver is restored.

Calcium gluconate given intravenously may be of value in the control of the pruritus but is not effective in the control of hemorrhage.

Prior to the development of vitamin K, a preoperative transfusion of blood was considered essential in jaundiced patients. The administration of normal blood served to raise the prothrombin above the critical level and was helpful in preventing hemorrhage. Transfusion before operation is no longer essential unless the patient is anemic but it should be remembered that blood transfusion is still the most rapid means available for raising the prothrombin level and correcting a hemorrhagic tendency.

OPERATIONS FOR CARCINOMA · OF THE PANCREAS

Operations for carcinoma of the pancreas can be divided into three groups—exploratory, palliative, and curative.

In the first group the abdomen is explored and the diagnosis is confirmed without any attempt being made to relieve the patient's symptoms.

Cholecystostomy is the least effective of the palliative operations. By the establishment of an external biliary fistula the jaundice and pruritus are relieved but no provision is made for the restoration of the bile to the intestinal tract. The patient continues to fail to assimilate nourishment and the course is rapidly downhill to a fatal termination. A hemorrhagic tendency often develops and the care of the biliary fistula is an additional burden to the patient and his relatives.

Cholecystogastrostomy or cholecystoduodenostomy are the best of the palliative operations for carcinoma of the pancreas. They have the advantage of reestablishing the continuity of the flow of bile and abolishing the annoying pruritus. Striking

subjective and objective improvements in the general condition of the patient often follow. Cases have been reported in

FIG. 2. First stage of radical resection of the head of the pancreas. Figure shows common duct ligated and gall-bladder anastomosed to the stomach.

which patients with proved carcinoma of the pancreas have lived nearly five years in relative comfort after this type of palliative operation. One patient in this series lived three years, eventually dying from carcinoma. This type of operation has the additional advantage of providing a permanent cure in the occasional case in which the obstruction of the common duct is caused by an inflammatory lesion rather than a carcinoma.

In this series the postoperative mortality rate was actually 7 per cent lower in the cases in which anastomoses between the biliary tract and the gastrointestinal tract were made than in the cases in which exploration only or exploration and cholecystostomy were performed. By reintroduction of the bile into the intestinal tract, the bleeding tendency was apparently overcome and late hemorrhage was avoided. The incidence of postoperative bleeding was only one-fourth as high in the patients subjected to anastomosing operations as in those who were merely explored or in whom only a cholecystostomy was done.

For these reasons, in treating patients with jaundice secondary to carcinoma of the pancreas, we prefer, whenever

FIG. 3. Second stage of radical resection of the head of the pancreas. Figure shows common duct and cholecystogastrostomy performed at the first stage. In addition, the stomach has been severed at the pylorus and the proximal end inverted. The duodenum and head of the pancreas have been removed and the distal end of the duodenum has been inverted. A posterior gastroenterostomy has been performed.

possible, to anastomose the biliary and intestinal tracts.

The third type of operation for carcinoma of the pancreas is the operation of choice in the early case. We are still handicapped by our failure to establish the diagnosis early enough to make this type of operation widely applicable. Yet autopsy statistics indicate that in approximately 10 per cent of the cases of carcinoma of the pancreas and in a considerably higher percentage of the cases of carcinoma of the bile ducts and ampulla of Vater the carcinoma has not metastasized at the time of the patient's death.[4]

The radical operation for carcinoma of the pancreas as described by Whipple[5] consists of a block resection of the duodenum, head of the pancreas, and adjacent fatty and areolar tissue. It is based on sound principles of cancer surgery and has already yielded encouraging results in a limited series of cases. Of the twelve cases

reported in the literature, six patients have survived the operation and one of these (reported by Whipple) is alive and

may result in the development of an ascending biliary infection. The second stage of the procedure will be facilitated

FIG. 4. Gross specimen of carcinoma of the pancreas surgically removed. Patient alive and well six months after operation.

well nearly three years after operation. I have recently reported a thirteenth case in which the patient is well six months following resection of the pancreas for carcinoma.* (Fig. 3.)

If the jaundice is of long duration and the liver function is impaired, the first stage (cholecystogastrostomy and ligation of the common duct, Fig. 2) should be performed under local anesthesia, using a splanchnic block. Since there is no great need for relaxation, the first stage of the operation can easily be performed under local anesthesia without subjecting a poor risk patient to unnecessary anesthetic hazards.

The cholecystogastrostomy stoma should be made an inch or more in diameter in order to assure the maintenance of an adequate stoma after the contraction of the gall-bladder. A stricture at this point

* Since the writing of this paper, the patient referred to has died as a result of contracture of the cholecystogastrostomy stoma. There was no gross evidence of metastasis or local recurrence of the tumor, but, microscopically, early carcinomatous change was noted in the epithelium of the bile ducts throughout the remaining portion of the pancreas. The stoma was over an inch in diameter at the time of operation, a fact which emphasizes the point that the stoma cannot be made too large.

at this time by ligation of the common duct with a black silk tie.

Exploration of the common duct and of the pancreas should always be performed at the time of the first stage. The duct is usually dilated to such a degree as to allow the insertion of the index finger and the obstructing lesion can be palpated from within the duct. Metastasis should be searched for and the degree of fixation of the tumor judged. A satisfactory method of palpating the head of the pancreas is by inserting the left hand behind the pancreas in the foramen of Winslow and palpating the organ bimanually, the right hand lying on the mesocolon over the head of the pancreas. The pancreas should not be exposed at this stage lest adhesions thus produced render the second stage more difficult.

The second operation (Fig. 4) should be performed as soon after the first as the patient's general condition permits, but a minimum of two weeks should be allowed for convalescence. Excellent exposure for this stage can be obtained through a transverse epigastric incision. A general anesthetic is recommended in order to insure adequate relaxation. Exposure of

the region of the pancreas is obtained by diversion of the gastrocolic omentum. The stomach is then divided at the pylorus and the proximal end closed. Silk is recommended as a suture material because if pancreatic juice should escape, catgut may be digested. The duodenum is next mobilized and the gastroduodenal artery ligated. No attempt is made to separate the duodenum from the pancreas, the dissection being carried along the lateral and posterior aspects of the duodenum. The duodenum is again divided distal to the head of the pancreas and its distal end closed. The hand can then be inserted behind the duodenum and the head of the pancreas and these structures are lifted forward in a block.

Palpation of the pancreas will now show the extent of the tumor. A blunt instrument can be inserted beneath the body of the pancreas well distal to the tumor and the pancreas can be divided. The pancreas itself is not very vascular but underlying it are the portal, splenic, and superior mesenteric veins which receive small tributaries from the pancreas and render this portion of the dissection difficult.

The cut end of the pancreas can best be ligated by three mattress sutures of stainless alloy steel wire passing through the entire pancreatic substance and with the central suture so placed as to encircle the duct completely. The external secretion of the pancreas is not essential to the life or health of the patient. The operation is completed by a posterior gastroenterostomy to reëstablish the continuity of the gastrointestinal tract. (Fig. 4.) The pancreatic bed should be drained for three or four days, two cigarette drains being sufficient for this purpose.

In Whipple's most recent publications he has favored a more elaborate cholecysto-intestinal anastomosis carried out on the Roux principle. By this means he hopes to decrease the incidence of postoperative cholangitis. It is my impression, however, that the greatest hazard is at the first stage when the patient is deeply jaundiced, poorly nourished, and in a critical condition. I therefore favor the simplest operation which will allow bile to enter the intestinal tract and I believe that if the cholecystogastrostomy stoma is made large enough to prevent formation of a stricture, cholangitis of a serious degree will not occur.

Palpation of pancreas containing a carcinoma at the head may give a mistaken impression as to the extensiveness of the lesion. The pancreatic duct is often completely occluded and proximal to the point of occlusion it is enormously dilated and filled with fluid. The pancreatic tissue is flattened to a shell around the tensely dilated duct and palpation of the pancreas suggests that the entire organ is hard and involved in the tumor. Careful palpation, however, reveals a difference in the stony hard consistency of the tumor in the head and of the tense slightly fluctuant body of the pancreas. If doubt exists, a small needle can be inserted into the duct and the fluid aspirated so that the true extent of the tumor can be better ascertained.

POSTOPERATIVE TREATMENT

Operations performed in the presence of jaundice carry with them the added risk of the development of liver failure. This risk varies directly with the duration of the jaundice and the degree of liver damage. It is reduced by avoiding a prolonged general anesthesia and by the preoperative administration of glucose solution and a high carbohydrate diet. The most critical period, however, comes after the operation and at this time, when the patient is unable to take food and fluids by mouth, it is most important to protect the liver by the intravenous administration of large quantities of glucose solution.

We have demonstrated clearly in hyperthyroidism that glucose given intravenously by the continuous drip method is much more effective in controlling the postoperative thyroid reaction than is the

intermittent administration of large quantities of glucose solution. When the glucose is given intermittently it is usually given too rapidly and a great deal of it is eliminated by the kidneys. The same applies in obstructive jaundice; if the maximum assimilation of the glucose is to be obtained and the liver is to be given maximum protection, the glucose should be given continuously.

In a recent case of obstructive jaundice the patient began to hiccough on the second postoperative day and on the third postoperative day he became confused and delirious. His condition appeared desperate, blood transfusion was given in order to prevent any tendency to bleed, but it was not until a continuous drip of glucose was given intravenously that the delirium cleared. The hiccoughs finally ceased, the liver again began to function, and the bile, which had been pale and had been excreted through the T-tube in very small amounts, rapidly returned to normal quality and quantity. This patient was practically afebrile during the period of his delirium and there was no evidence of pulmonary or intraperitoneal complication. The toxic reaction was certainly the result of liver failure and the dramatic recovery was in all probability directly attributable to the administration of large quantities of glucose intravenously and continuously.

A second complication of surgery in the biliary tract is the development of cholangitis, a complication which has occurred, although without fatal results, following 29 per cent of the operations in which anastomoses were made between the biliary tract and the gastrointestinal tract. Hitherto we have had no logical means by which to combat this type of infection.

We have demonstrated in the experimental laboratory that sulfanilamide is excreted in the bile. External biliary fistulae were made in dogs, large doses of sulfanilamide were given to these animals, and we were able to recover sulfanilamide in the bile in amounts equivalent to those present in the blood. Clinically, we have tried sulfanilamide in several cases of cholangitis and results have been promising. In one case an anastomosis between the gall-bladder and the jejunum had been performed, a cholecystoduodenostomy or cholecystogastrostomy being rendered impossible by an extensive carcinoma of the stomach which had invaded the common duct and resulted in jaundice. After operation, as is often the case when the lower portion of the intestinal tract is anastomosed to the biliary system, severe cholangitis with chills, fever, and jaundice developed. The patient was treated with sulfanilamide and within forty-eight hours the chills and fever had subsided and the temperature fell to normal levels. We have since observed similar responses to sulfanilamide in other patients with cholangitis. I therefore believe that sulfanilamide offers a definite promise of being as valuable an antiseptic in the biliary tract as it has proved to be in certain stubborn infections of the urinary tract.

A third troublesome postoperative complication following operations on jaundiced patients has been pneumonia. In many instances the pneumonia begins with a patchy atelectasis. Spinal anesthesia used in operations on the upper abdomen appears to increase the patient's susceptibility to this complication and local anesthesia diminishes the risk to a minimum. Following operation the patient should be instructed in periodic deep breathing exercises, the position should be changed frequently, and the usual wide adhesive support of the dressing should be dispensed with, the dressing being covered by a single longitudinal strip of adhesive which will have no tendency to limit the respiratory movements.

SUMMARY

1. With the advent of vitamin K for the control of the hemorrhagic diathesis in jaundice, with the use of the continuous drip of glucose in the prophylaxis and treatment of postoperative liver failure,

and with the prospect that sulfanilamide may be of value in the control of cholangitis, it is probable that there will be a lowering of the surgical mortality in patients with obstructive jaundice and an increasing interest in the surgical treatment of carcinoma of the head of the pancreas.

2. Twelve cases of radical resection of the head of the pancreas have already been reported; six patients have survived operation, and one of these patients (reported by Whipple) is still alive and well nearly three years after the original operation.

3. The greatest progress in the surgical attack on carcinoma of the pancreas will come when physicians and surgeons recognize that carcinoma of the pancreas is a curable disease provided the diagnosis is established before the tumor has invaded vital structures.

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SARCOMAS OF THE SMALL INTESTINE*

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INTRODUCTION

OF the tumors found in the small intestine, sarcomas are the most frequently reported. Of the 11,000 autopsies performed at the Cook County Hospital from 1929 to May, 1938, 183 were malignant tumors of the intestine, ten of which were found in the small intestine. Seven of these ten cases were carcinomas, one a malignant argentaffinoma, and two lymphosarcomas. Of the surgical and biopsy specimens of the intestine, five cases of the small intestine were encountered four of which were carcinomas and one a leiomyosarcoma.

The above figures include the three cases to be reported in this paper.

The most frequently described sarcoma of the small intestine is the lymphosarcoma, particularly the reticulum cell type. In 1919, Graves[1] reviewed the literature and collected a total of 249 sarcomas of the intestine. In 1932, Ullman and Abeshouse[2] added 126 cases, including one of their own. Since then cases have been reported by Raiford,[3] Magnusson,[4] Geschickter,[5] Mayo and Robbins,[6] Joyce,[7] Leveuf and Godard,[8] and others.

Of the more infrequent types of sarcomas found in the small intestine, leiomyosarcoma, fibrosarcoma, spindle cell sarcoma and giant cell tumors have been reported in the literature by Nickerson and Williams,[9] Goldsmith,[10] Glass and Oldberg,[11] Geschickter,[5] Baltzar,[12] Koch,[13] Gohn and Hintz,[14] Van Knowe,[15] Libman,[16] Magnusson,[4] Renvall,[17] Bjorkenheim,[18] McDermott,[19] and others.

We have the opportunity of reporting in addition to two cases of lymphosarcoma a case of leiomyosarcoma of the small intestine which was removed at operation and submitted for examination to the surgical pathology laboratory.

REPORT OF CASES

CASE I. A white man, 50 years old, was apparently perfectly well until four weeks prior to his entrance into the hospital. At that time he experienced anorexia, his skin turned yellow, he suffered dull, aching pains in the epigastrium, usually one hour after meals. At the time of his admission to the hospital, the pain was radiating in character and was aggravated by the ingestion of fatty and fried foods. On the four days prior to admission, he noticed black stools. Chills and fever were present for ten days, and in the previous four weeks he had lost 20 pounds.

The patient's temperature was 101°F., his pulse 90, respiration 24, and blood pressure 110 systolic and 70 diastolic. He was markedly jaundiced. A few râles were heard in both bases. The abdomen was greatly distended and elicited a distinct fluid wave. The liver was palpated four fingers below the right costal arch, and its edge was irregular and nodular. The spleen was enlarged and firm on palpation.

On the basis of these clinical findings a diagnosis of a primary carcinoma of the pancreas or extrahepatic bile ducts with extensive metastases to the liver was made. Bile was found in the urine. The patient died two days after admission to the hospital, and a detailed laboratory work-up could not be done.

Autopsy showed a well developed white male, 181 cm. long, and 72 kilograms in weight. The skin was yellow green in color and the sclerae were yellow gray. The abdomen was covered diffusely with scratch marks. The abdominal cavity contained 150 c.c. of a blood-tinged fluid. The surface of the liver was covered by loosely adherent blood clots.

The stomach was markedly distended and filled with coffee brown fluid. The mucosa was light brownish gray and the folds were distinct. In the second portion of the duodenum there was a firm cauliflower-like mass measuring 60 by 45 mm. The surface of this mass

was yellowish-brown-gray, and in the center there was a deep ulcer measuring 30 by 40 mm. covered by a dirty greenish-gray membrane.
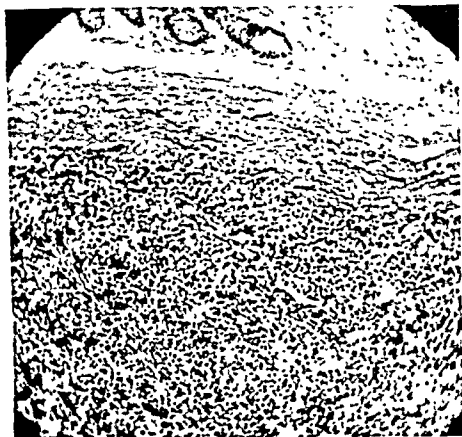
FIG. 1. Case 1. × 72. Hemalum and eosin stain. Showing diffuse infiltration of the lymphoid cells into the muscularis propria.

On tracing the common duct from the liver, it was found to empty into the duodenum on the superior margin of the ulcer. The lumen was occluded at its tip, and the ampulla was replaced by the dirty greenish-gray to yellowish-gray tumor tissue. Beyond this obstruction the common duct was dilated to a circumference of 31 mm. The remainder of the mucosa of the small intestine was smooth and grayish-tan in color. The large intestine was smooth except that in the cecum there were four isolated, freely movable sessile nodules, the largest being 15 by 5 mm. in diameter and of a purple-grayish-tan in color.

The liver weighed 4,250 Gm. Its surface was profusely studded with grayish white nodules which had umbilicated centers and measured up to 8 mm. in diameter. The sectioned surface of the liver was grayish-green mottled with circumscribed areas of purple-gray. The markings of the liver lobules were distinct. The bile ducts were very much dilated and contained thick, viscid bile. The gallbladder was distended and also contained bile of a similar appearance.

The pancreas measured 16 by 50 by 40 mm. The pancreatic duct was dilated to a diameter of 3 mm. and filled with a thick mucoid material. The duct as it approached the duodenum became more and more dilated and emptied alongside the common duct at the superior margin of the ulcerated tumor mass. The mouth of this duct was compressed by this greenish

to yellowish gray tissue. Throughout the body of the pancreas were found opaque yellowish gray white nodules. The body of the pancreas,
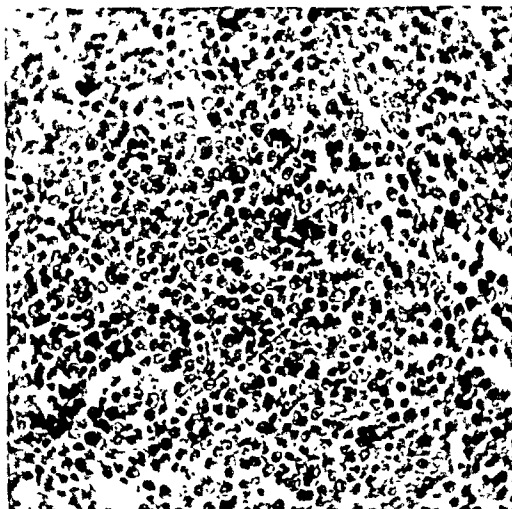
FIG. 2. Case 11. × 144. Hemalum and eosin stain. Showing the diffuse infiltration of the tumor cells into the wall of the intestine.

the left adrenal, and the upper pole of the left kidney were firmly bound down to each other by yellowish-gray to purplish-gray tissue.

The periaortic lymph nodes were enlarged up to 40 mm. in diameter and when sectioned were light yellowish-gray to purplish-gray in color.

Microscopic Findings. Histologic studies were made from different levels of the tumor in the duodenum, from the liver, the peripancreatic lymph nodes, the pancreas, the kidneys, and the nodes of the intestine in the region of the cecum. The tissues were stained with hemalum eosin, Bielschowsky's silver stain, Heidenheim iron hematoxylin, and iron carmine stain.

Sections taken through the ulcerated area of the duodenum revealed the surface of the ulcer to be composed of a layer of necrotic tissue in which pink staining residue of cells was seen. These cells were enmeshed in fibrin. At the margin of the ulcer, the duodenal mucosa was well preserved. In this area the submucosa up to the muscularis mucosa was infiltrated by eosinophiles, leucocytes, and plasma cells. Beneath the muscularis mucosa were numerous Brunner glands which were widely separated from each other by large numbers of deeply staining lymphoid cells with round, oval, or slightly indented nuclei, many of them containing distinct nucleoli.

The chromatin granules were coarse and deposited chiefly in the periphery of the nucleus to be connected with each other by fine, broken chromatin threads.

The cytoplasm was acidophilic, usually small in amount, and surrounded by a distinct cell membrane. The cells separated widely and often replaced the fibers of the muscularis propria (Fig. 1) and extended into the fat tissue of the subserosa. Beneath the ulcerated area described above, small areas of necrosis sometimes were seen associated with hemorrhage and focal areas of fibrosis. There were occasional multinucleated cells with nuclei closely resembling those of the lymphoid cell described above. Atypical mitotic figures were frequently found. The blood capillaries and venules were dilated, and in some venules small accumulations of tumor cells were seen.

A section of the ulcer taken through the area where the common duct and pancreatic duct enter into the duodenum showed a diffuse ulceration of their mucous lining with the formation of a necrotic membrane. Beneath this membrane were large groups of lymphoid cells similar to those described above, extending into the wall of the pancreatic and common duct. Silver stains revealed fine fibrils surrounding the individual cells of the tumor.

The microscopic examination of the liver disclosed that in many places the sinusoids were markedly dilated and filled with lymphoid cells of the type described in the duodenum. The liver cords were atrophic. In places large portions of the liver parenchyma were replaced by lymphoid cells between which numerous multinucleated cells were seen. The adjacent liver parenchyma was compressed, and many of the liver cells were stuffed with a brownish-green pigment. Small bile casts were seen in the intercellular bile capillaries. The Kupffer cells were swollen and iron carmine stain showed them to be stuffed with blue staining iron granules. The periportal areas showed a moderate infiltration of round cells, and in places the blood vessels were occluded by tumor thrombi.

The normal architecture of the peripancreatic lymph node was completely destroyed by large numbers of lymphoid cells and many multinucleated cells similar to those described in the tumor mass of the duodenum. The capsule of the lymph node and the adjacent fatty tissue were infiltrated by these tumor cells. Throughout the lymph node the blood vessels were dilated and thin-walled.

In sections of the pancreas the lobules were found to be well preserved, but the perilobular and interlobular connective tissue and fat tissue were diffusely infiltrated by the lymphoid cells and plasma cells. Here and there small islands of fat tissue contained bluish staining calcium material.

Anatomic Diagnosis. Lymphosarcoma (reticulum cell type) of the duodenum, involving the ampulla of Vater, with extension to the tissue about the left adrenal, left kidney, and the pancreas. Metastases to the liver, to the periaortic, and peripancreatic lymph nodes. Also: (1) bronchopneumonia in the right lower pulmonary lobe; (2) syphilitic aortitis and moderate sclerosis of the coronary artery; (3) moderate ascites, icterus gravis, nodose goiter, multiple polyps of the cecum and focal fat necrosis of the pancreas.

CASE II. A white man, aged 42, entered the Cook County Hospital on July 7, 1936 with a complaint of abdominal pain for the previous one and one-half years, associated with frequent vomiting. The vomitus was occasionally coffee-ground in color. A weight loss of 30 pounds occurred during the course of his illness.

The patient's skin was slightly icteric. The temperature was 105 degrees, pulse 100, respirations 24, and the blood pressure 108 systolic and 50 diastolic. Small lymph nodes were palpable in the supraclavicular region. The lung examination revealed no abnormal findings. A systolic murmur over the apex of the heart was heard. The liver was enlarged two fingers below the level of the chest. X-ray examination of the gastrointestinal tract was negative.

The red blood cell count was 3,370,000 and the white cell count was 7,400 with 84 per cent polymorphonuclear leucocytes. The hemoglobin was 50 per cent on admission. An examination of the gastric juice after an Ewald's meal revealed 64 units of free hydrochloric acid. Fecal examination was repeatedly negative for blood. Electrocardiogram readings were normal. Wassermann tests were negative. Clinically the patient offered a symptom complex which was difficult to interpret. He was placed under observation, and after a time it was considered advisable to explore the abdomen surgically for a possible carcinoma

of the pancreas. At this time there was much tenderness over the entire abdomen, and a questionable mass was palpated in the right upper quadrant.

At operation the small intestine and mesentery were found to be covered with tumor tissue. The involvement was too extensive for resection, and only a biopsy was taken for diagnosis. A section of the biopsy revealed a lymphosarcoma. Postoperatively the patient developed a marked ascites and pitting edema of the lower extremities. Four and one-half weeks after the operation he expired.

Autopsy Findings. The patient was a poorly nourished white man whose skin was dirty gray-brown in color. The superficial lymph nodes were not enlarged. The lower half of the abdomen was slightly distended, and near the midline below the right costal arch was a linear scar 13 cm. in length. There was distinct clubbing of the fingers and toes.

The midline fat was absent. The abdominal cavity contained 1,800 c.c. of an opaque light yellow gray fluid. A loop of small intestine and greater omentum was adherent to the anterior abdominal wall in the region of the scar. The greater omentum contained moderately firm, light pinkish gray plaques measuring up to 4 by 2 by 1 cm. in diameter. Similar though larger plaques were found in the gastrocolic ligaments. In the mesentery of the small intestine, near the root, were several moderately firm, light pinkish gray nodes, the largest 3 by 1 by 2 cm. in diameter. The loops of the small intestine were markedly distended by gas and in places adherent to each other.

Shining through the serosa were many firm, lobulated, grayish nodes, which often involved the entire circumference and extended into the adjacent mesentery. The largest of these nodes measured 11 cm. in longitudinal and 6.5 cm. in transverse diameter.

The mucosa of the stomach was light pinkish-tan. The folds were distinct. Twenty mm. from the pyloric ring, the posterior and lateral wall of the first portion of the duodenum showed an ulcerated oval plaque measuring 5 by 3 cm. in its greatest dimension. The ulcerated area was kidney-shaped and exposed a light grayish-tan and moderately firm tissue. The lower portion of the plaque extended into the second portion of the duodenum. In the third portion of the duodenum the wall was thickened up to 20 mm., was gray-white and

moderately firm, and contained an 18 by 10 mm. bean-shaped ulcerated area.

The uppermost portion of the jejunum was twisted upon itself, the wall was infiltrated by numerous moderately firm, pale purplish-tan and centrally ulcerated, discrete, and confluent plaques which involved all layers of the wall, obscuring their differentiation. The largest plaques measured 4 by 3 cm. in diameter and were raised for about 8 mm. These plaques continued throughout the jejunum and in places were fused to form large solid masses which completely surrounded the lumen of the intestine, without constricting it. The circumference of the intestine in that region measured 95 mm. There were multiple irregular ulcers exposing dirty, light yellowish-gray tissue. In the mid-portion of the ileum the changes became gradually less extensive, but single ulcerated plaques were found down to the region of the ileocecal valve. The mucosa of the large intestine was light purplish-brown. The lumen was filled with thick, pasty brown material. The mucosa of the rectum was light purplish-brown.

The adrenals were flattened by moderately firm, light purple-pink infiltrations of the periadrenal tissue, which was up to 16 mm. thick and was sharply separated from the light yellow-gray of the adrenal cortex. The cortex was about 1 mm. thick. The inner zone was dark brown.

Microscopic Findings. Microscopic studies of this case included the intestine, pancreas, adrenals, spleen, and liver. Azan, hemalum and eosin stain, and silver impregnation were used.

The small intestine in the region of the tumor wall was diffusely infiltrated by large round cells which separated and replaced the preëxistent structures. This infiltration was most extensive in the submucosa and muscularis propria but also affected the subserosa, as a rule. Groups of muscle fibers of the muscularis propria could be encountered between the tumor cells. The infiltration often stopped at the muscularis mucosae, and in these places the mucosa appeared to be well preserved, except for a slight edematous loosening of the stratum proprium. There were, however, areas in which the tumor cells penetrated the muscularis mucosae and spread into the mucosa. They filled and expanded the

villi, while the epithelial lining of the crypts remained intact. Later, the thickened, densely infiltrated villi seemed to fuse together. The

FIG. 3. Case III. × 276. Hemalum and eosin stain. Showing many multinucleated cells and the tendency of the cells to form syncytia. There is a marked infiltration of round cells and polymorphonuclear leucocytes.

crypts disappeared, and the entire thickness of the wall appeared as a solid and uniform mass of tumor tissue bordering at the lumen as well as reaching the serosa. (Fig. 2.)

The tumor cells varied slightly in size and shape. They were round or short oval and were from three to four times the size of small lymphocytes. They showed a distinct rim of homogeneous cytoplasm. Most of the nuclei were round, some oval, and a few bean-shaped. The nuclear membrane was distinct and the chromatin formed coarse granules connected by thinner bridges. There were two or three round nucleoli. Mitoses were very numerous.

In the Azan-stained sections the tumor cells appeared to be arranged in small nests encircled by bright blue fibers of moderate thickness. The blue fibrils often extended between the cells. The coarse fibers carried thin-walled capillaries. The wall of the larger vein was frequently infiltrated by tumor cells which protruded into the lumen in the form of small polyps or undermined the endothelium for a considerable distance.

After silver impregnation the coarser bundles between the cell nests appeared as wavy black strands from which single fine fibrils broke off to extend between the cells. These latter fibrils, too, were distinctly tortuous.

The periadrenal fat tissue was densely infiltrated by tumor cells which resembled those in the intestine. The tumor cells extended close to the capsule of the adrenal cortex but stopped at this point and did not continue into the capsule and cortex. The cortex was well preserved and contained a moderate amount of lipoid material.

The tissue about the pancreas appeared as a uniform mass of tumor cells which often continued into the pancreas following the interlobular septa. The latter thus became much thickened. Here and there the tumor cells also broke into the lobules and replaced the parenchyma, which, under the influence of the invading cells, seemed to melt away. In the remaining portions of the pancreas the acini and islands were intact.

Anatomic Diagnosis. Reticulosarcomatosis of the small intestine extending from the first portion of the duodenum to the ileocecal valve. Sarcomatous infiltrations of the mesenteric, peripancreatic and periadrenal fat tissue. Also: (1) chylous ascites and hydrothorax of the right side; (2) brown atrophy of the liver and myocardium; (3) sand-like uric acid concretions in both renal pelves; (4) focal bronchopneumonia of the right upper pulmonary lobe; (5) atrophy of the thyroid.

CASE III. A white woman, aged 38, was admitted to the hospital on May 6, 1937 with complaints of abdominal pain and vomiting for five days. Constipation was present since 1933.

On examination, the patient did not appear acutely ill. Her temperature was 98°F., pulse 118, respirations 20, and the blood pressure 100 systolic and 80 diastolic. Head, neck, and chest examinations were essentially negative. The abdomen was scaphoid. The liver, kidneys, and spleen were not palpable. Tenderness, however, was elicited in the epigastrium and about the umbilicus.

Laboratory examination revealed blood in the stools. The urine examination was essentially negative. The white cell count varied on different occasions from 14,950 to 13,600.

The patient had no bowel movement for two days, and an emesis suggested fecal material. A diagnosis of intestinal obstruction was made, and the patient was transferred to a surgical ward. At operation a tumor in the jejunum was found attached to the mucosa along the mesenteric border. The tumor was obstructing the lumen of the intestine and was associated with an intussusception at the ileojejunal junction. The tumor was excised, and an end-to-end anastomosis was made. Recovery was uneventful.

TABLE I
MALIGNANCIES OF SMALL AND LARGE INTESTINE
1929–1938

		Small Intestine				Large Intestine										
Year	P M or Surg	Duodenum	Jejunum	Ileum	Total	Cecum	Ascending Colon	Hepatic Flexure	Transverse Colon	Splenic Flexure	Descending Colon	Sigmoid	Rectum	Anus	Unclassified	Total
1929	P M	A-1 E-1	F-1		3	A-1				A-2 B-1		A-4 B-1	A-2 C-1 D-1			13
	Surg														8	8
1930	P M					A-1	A-1	C-1				A-5 B-1	A-6 B-1			16
	Surg												A-7 B-1			8
1931	P M		A-1		1	A-1		A-1	A-1			A-2	A-4 B-1			10
	Surg					A-1							A-21 D-4			26
1932	P M					A-3	A-1 B-1			A-1	A-2	A-2 B-1	A-4			15
	Surg					A-6							A-34 D-7			47
1933	P M	A-1	A-2		3	A-2 B-1		A-1 B-2				A-4 C-1	A-7 D-2			20
	Surg		A-1		1	A-13							A-44 D-1			58
1934	P M					A-2 E-1	A-1	A-1	A-2	A-2	A-4	A-8 B-1 C-1	A-10 B-3 D-1	E-1		38
	Surg							A-1 E-1	A-1		A-1	A-3 B-1	A-42 B-3 C-1 D-6			60
1935	P M		A-1		1						A-3	A-6	A-3 B-1			13
	Surg		A-2		2	A-4	A-1	A-2 B-1	A-1	A-1		A-9	A-48	A-1		68
1936	P M	G-1		G-1	2	A-3			A-2 E-1	A-1		B-3	A-8			18
	Surg					A-2		A-1	A-1 B-1	A-1		A-8	A-58 B-5 C-2	B-1		80
1937	P M					B-1	A-1	E-1	A-1		A-1	A-6 E-2	A-12 B-1			26
	Surg			H-1	1	A-3		A-2	A-1 B-2	A-1		A-14	A-65 B-8 C-2	B-1 D-1		100
1938	P M												A-4			4
	Surg			A-1	1				·			A-4	A-17 B-1	D-1		23
Total		4	8	3	15	45	6	15	14	10	11	87	449	8		

Key A—Adenocarcinoma
    B—Colloid carcinoma
    C—Scirrhous carcinoma
    D—Squamous cell carcinoma
    E—Medullary carcinoma
    F—Argentaffinoma
    G—Lymphosarcoma of the reticulum cell type
    H—Leiomyosarcoma

Surgical Pathology Report. The specimen consisted of a piece of jejunum 2 cm. long. Extending into and completely obstructing the lumen was a firm mass measuring 3 by 2.5 cm.

in diameter. This mass was attached to the mesenteric portion of the mucosa by a broad base measuring 2.5 cm. On sectioning, the tumor was light grayish-yellow, mottled with small purple areas up to 0.5 cm. in diameter.

Microscopic Description. Sections of the tumor were stained with hemalum and eosin after the Van Gieson method, and with Mallory phosphotungstic acid hemotoxylin.

The mucosa overlying the node was missing, and the free surface was composed of a necrotic membrane infiltrated by numerous polymorphonuclear leucocytes. Beneath this membrane the tissue was composed of interlacing bundles of elongated and tortuous cells containing large and oval nuclei with coarse chromatin granules scattered irregularly throughout. Many of the nuclei contained distinct nucleoli. The cytoplasm was acidophilic and surrounded by an indistinct membrane which, in places, seemed to be continuous with the adjacent cellular membrane of another cell. (Fig. 3.) In addition to this type of cell, numerous multinucleated giant cells were seen. Stained after Van Gieson, these cells appeared yellowish-brown, surrounded by fine, pale pink connective tissue fibrils. In sections stained with Mallory's phosphotungstic acid hemotoxylin, the cells contained fine longitudinal fibrils in their cytoplasm. These were also present in some of the multinucleated giant cells. The bundles of cells were widely separated by accumulations of neutrophilic and eosinophilic leucocytes. The blood vessels were markedly dilated and thin-walled.

The diagnosis of a leiomyosarcoma was made.

DISCUSSION

In our study of the autopsy material at the Cook County Hospital from 1929 to 1938 (Table I), we found 183 malignancies of the large and small intestine. Of these 173 were located in the large bowel and ten in the small bowel. In the larger group, the average age for females was 53.95 years and for males 55.47 years. The youngest female was 28 years, the youngest male 16 years. The oldest female was 80 years, and the oldest male the same. Of the malignancies of the small bowel, the average age for females was 46 years and for males 52 years. The youngest female was 42 years, the youngest male 36 years. The oldest

female was 49 years, the oldest male 77 years.

In our series, there were 129 males and fifty-four females. There were 154 white patients and twenty-nine colored.

In our surgical series from 1930 to May, 1938, excluding 12,705 appendectomies, 1,560 operations and biopsies were performed on the large and small bowel. In this series, 483 malignancies were found. Only five were located in the small intestine. Of the 478 malignancies of the large bowel the average age for females was 54.62 years and for males 55.24 years. The youngest female was 30 years, the youngest male 20 years. The oldest female was 81 years, the oldest male 79 years. In the five malignancies of the small bowel, the age and race were not obtainable. The age of twenty females and sixty-seven males in the group of large bowel malignancies was not obtainable; our age average was determined from the remainder of the 396 cases.

Lymphosarcoma for some time was frequently confused with Hodgkin's disease, leucosarcoma, and lymphatic leucemia until Kundrat[20] first differentiated it from the group of closely allied diseases of the lymphatics. He describes the lymphosarcoma as a tumor arising only from a group of lymph nodes and not from a single node. Occasionally it may arise in the lymphoid tissue of the mucous membrane from which it may extend to the neighboring lymph nodes and the surrounding tissue, as is well demonstrated in two of the cases reported here. He segregates these tumors from true neoplasms because they are not spontaneous growths of multiple origin with extension by metastases, but a regional disease of lymphoid tissue which propagates itself through the lymph paths. However, it has since been shown that distant metastases have occurred. (Ewing,[21] Libman,[16] Koch,[13] Graves,[1] Ullman and Abeshouse.[2])

Two examples of distant metastases are demonstrated in the cases of lymphosarcomas described in this paper. In the first case distant metastases were found in the liver and sinusoids of the liver lobule.

In the second case the tumor was found in the periaortic lymph nodes, and it had also invaded the lumen of the blood vessels, as well as the wall of the blood vessels. We feel, therefore, that these tumors should not be segregated from the other types of malignant neoplasms, since the lymphosarcomas possess the same potentialities of dissemination by way of the blood stream to the distant parts of the body. It cannot be denied, however, that the greatest growth is local, with marked invasion of the adjacent organs.

The recognition of a lymphosarcoma from a histologic examination of the tumor tissue may lead to a good deal of confusion when one considers the possibility of an existent Hodgkin's disease, leucemia, or intestinal tuberculosis. It is further confusing since the true tumor may possess a single type or all varients of tumor cell found in a true case of lymphosarcoma. Lubarsch and Henke[22] describe eight different cell types: the lymphocytic type; the lymphoblastic type; the reticulum cell type, composed of fine cytoplasmic processes, forming a network in which are found other cellular elements such as lymphocytes and lymphoblasts, although at times the network is so dense that there is no room for other cells and the tissue resembles protoplasmic glia; the dwarf cell type; the giant cell type; cells of phagocytic structure, composed of honeycombed cytoplasmic structure in which products of ingestion are frequently seen; the plasma cell type; and the plasmomast cell type.

The two cases reported here are of the reticulum cell type which is comparable with the lymphoblast type described above.

That these tumors occur only in one portion of the intestine or diffusely throughout the small intestine and at times involve the large intestine has been frequently shown. A case of each type is represented here. The focal types are more malignant than the diffuse type,[21] although both types may prove rapidly fatal.

The third case in this report deals with a leiomyosarcoma. Histologic examination, especially with Mallory phosphotungstic acid hemotoxylin, discloses the longitudinal myofibrils in the bundles of tumor muscle cells, and even in some of the multinucleated giant cells. The origin of this type of tumor may be from either the muscularis mucosa or the muscularis propria. This tumor is infrequently found in the small intestine. The ratio of myosarcomas to other types of tumors in the small intestine is 2 to 63, according to Corner and Fairbanks,[23] 1 to 96, according to Speese.[24]

The pathogenetic basis of tumors of the intestinal tract is far from being definite, although many suggestions have been made regarding their origin. Lymphosarcomas of the intestine have been regarded by some authors to be due to infection, particularly tuberculosis, syphilis, or typhoid fever (Joyce,[7] Ligman,[16] Nothnagel,[25] Birch-Hirshfield,[26] and others). Ribbert[27] believes that these tumors are produced by aberrant, undifferentiated cells in which tuberculosis is purely accidental. In our cases no evidence of a latent or active tuberculosis was found at post-mortem examination. The clinical symptoms also were more in keeping with a diagnosis of malignancy rather than one of tuberculosis.

It is interesting to note in both cases of lymphosarcoma reported here that there was a pronounced absence of symptoms referable to intestinal obstruction. This is readily explained by the nature of the tumor growth—it grows peripherally and excavates the lumen of the intestine by ulceration, sometimes causing aneurysmal dilatation of the intestine. This finding is in keeping with those of other authors, although Liu[29] and Miller[30] have shown that obstruction is more common either by constriction of the lumen or by intussusception. Sarcomas are more frequent in the small intestine than are carcinomas, according to reports in the literature.[1,3] In our series, however, sarcoma was found to constitute only 20 per cent of all tumors of the small bowel. Nickerson and Williams[9] similarly report two sarcomas in ten cases of small intestinal tumors.

The other types of sarcomas found in the small intestine, chiefly the myogenic and

fibrosarcomas, usually offer early symptoms of intestinal obstruction since they have a tendecy to become pedunculated.

Of all the malignancies of the small intestine, the lymphosarcomas are the most rapidly growing, spreading early to the adjacent lymph nodes and later in the course of the disease to distant organs. Attempts at operative removal usually prove unsuccessful, since local tumor growths have recurrences and diffuse tumors are more or less inoperable. Liu,[29] however, has reported a number of cases in which patients have lived four years after resection of the tumor.

X-ray therapy should be given cautiously since the marked sensitivity of the mucosa to the x-rays may cause a secondary ulcerative and suppurative enteritis.

The myogenic tumors, due to their tendency to metastasize late, offer a good prognosis at early removal, although recurrences have been noted.

SUMMARY

Three cases of sarcoma of the small intestine are reported, two lymphosarcomas and one leiomyosarcoma, the former growing rapidly and spreading early to the adjacent lymph nodes, and the latter growing and metastasizing late. The lymphosarcomas are at times difficult to diagnose and may be confused with lymphatic leucemia, leucosarcomas, or Hodgkin's disease. In our series of cases, the number of sarcomas of the small intestine is much lower than the number of carcinomas.*

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* Since the submission of this manuscript, we have observed four additional malignancies of the small intestine in the surgical pathology laboratory of the Cook County Hospital. One was an annular constricting adenocarcinoma of the ileum; two were stenosing lymphosarcomas, one in the ileum and the other in the jejunum; the fourth appeared clinically to be a ruptured viscus, but proved to be a perforated ulcerating lymphosarcoma of the ileum. All of these were in males from 50 to 56 years old.

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IMMEDIATE SURGICAL VERSUS EXPECTANT MEDICAL TREATMENT OF CLINICALLY ACUTE GALL-BLADDER DISEASE*

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THE treatment of acute inflammation of the gall-bladder is a much debated problem in medicine today. Until recently, the expectant treatment was the routine procedure and only when the findings indicated a very severe suppurative process or when symptoms persisted after medical therapy, was drainage of the gall-bladder instituted. Cholecystectomy, however, was not considered safe treatment for acute gall-bladder disease. The expectant medical approach was justified on the theory that an acutely inflamed gall-bladder (in contradistinction to an acutely inflamed appendix) seldom perforated and that the danger to life from such a complication was not very great. Cholecystectomy was condemned as an operation which, at the acute stage, was considered more dangerous than cholecystostomy.

This conservatism is reflected in the report of Branch and Zollinger[2] who insist that only when cholecystitis is complicated by peritonitis should the patient be subjected to a surgical operation. To substantiate this they review 235 cases of acute cholecystitis only thirty-four of which were treated by immediate operation with a mortality of 20.5 per cent. On the other hand, among 195 patients whose operations were delayed, the mortality was only 8.7 per cent. Lahey[13] states that he has never seen an acute perforation of an inflamed gall-bladder which was not walled off. Federov[5] feels that surgical intervention during the acute stage involves much greater danger than in the chronic stage. Behrend[1] finds the mortality very high following operation in acute gall-bladder disease. Hartel[8] definitely prefers the conservative treatment.

On the other hand, Pfeiffer,[15] Robinson,[16] and Judd and Phillips[11] feel that during the acute stage operative intervention is definitely indicated. Among the more recently published articles, many point out that acute gall-bladder inflammation often leads to complications which seriously endanger the life of the patient. Wolfson and Rothenberg[21] reported thirty-one cases of acute non-calculous cholecystitis in six of which perforation occurred. In a large group observed and operated on by Heuer,[9,10] the incidence of gangrene or perforation was between 20 and 23 per cent. Taylor[20] thinks that much depends on the interval between the onset of the acute attack and the time of operation. Among 102 patients with acute gall-bladder disease, he found a mortality of 5 per cent in those subjected to operation within four days of the onset of the attack, while the death rate was 23.8 per cent where operation occurred later than the fourth day. Gray[6] cites the experience of some observers that results were much poorer when operation took place more than two weeks after the onset of the attack.

If the surgeon decides to operate during the acute stage, he has the choice of two procedures: cholecystectomy and cholecystostomy. A report of 200 operations performed by Steinke[17] in the acute stage shows fifteen deaths among 159 cholecystectomies, a mortality of 9.4 per cent. Of forty-one patients treated by cholecystos-

tomy, eight died—a death rate of 19.5 per cent. This suggests that cholecystectomy is a safer procedure during the acute phase of the illness. Many other authorities are in accord with this. Coughlin,[3] Heuer,[9] Lipschutz,[14] Judd and Phillips,[12] and Stone and Owings,[18] all prefer cholecystectomy for acute gall-bladder inflammation.

To secure further information on this question, we analyzed the data of the Newark (New Jersey) Beth Israel Hospital for the years 1933 to 1936. All charts filed under a diagnosis indicating an acute inflammatory condition of the biliary passages were surveyed. There were 101 cases of this nature. Table I summarizes the cases with reference to diagnosis and treatment. Table II lists only cases which, on operation, were shown to be acute inflammation of the biliary passages. This table excludes medically treated cases as well as those which were found to be non-acute. In this way, it was easier to evaluate results of surgical treatment in unquestionably acute cases. Table III represents a follow-up of thirty-seven cases that were either medically treated, or treated surgically without removal of the gall-bladder.

The charts show that the clinical symptoms (e.g., appearance of patient, tenderness in right upper quadrant, palpable gall-bladder, abdominal rigidity, fever, and leucocytosis) do not necessarily indicate the severity, acuteness or extent of the disease as it was found at operation. One could never tell how extensive or intense the inflammation was until the abdomen had been surgically explored. Acute cholecystitis may mislead, just as does acute appendicitis when, with seemingly minor symptoms, a perforation occurs. Some severe cases of cholecystitis, pericholecystitis, or perforation of the gall-bladder (with or without adhesions) showed such trivial symptoms that the physician believed medical treatment to be justified if not desirable. The data in the literature stress this hazard. Touroff[19] and notably Heuer[10] have emphasized the treachery of acute cholecystitis. When a

TABLE I

Type of Cases	Number	Notes
Operated, acute cholecystitis, ruptured gall-bladder, or empyema of gall-bladder. Cholecystostomy. Died.	10	One not proved to be acute.
Operated, acute cholecystitis or ruptured gall-bladder, or empyema of gall-bladder. Cholecystostomy. Lived.	17	Four not proved to be acute.
Operated, acute cholecystitis, ruptured gall-bladder, or empyema of gall bladder. Cholecystectomy. Died.	4	One not proved to be acute.
Operated, acute cholecystitis, ruptured gall-bladder, or empyema of gall-bladder. Cholecystectomy. Lived.	39	13 not proved to be acute.
Medical treatment only. Acute cholecystitis or empyema of gall-bladder. Died.	2	
Medical treatment only. Acute cholecystitis or empyema of gall-bladder. Lived.	21	2 refused operation. 2 left hospital against advice. 1 rehospitalized for cholecystostomy.
Medical treatment only. Acute hepatitis and cholecystitis. Lived.	1	
Partial cholecystectomy. Lived.	2	
Medical treatment only. Hepatic calculi, cholangitis. Lived.	1	
Medical treatment only. Cholangiitis. Lived.	1	
Operated. Empyema of gall-bladder and carcinoma. Incision and drainage of gall-bladder abscess. Died.	1	
Exploratory freeing of adhesions. Lived.	1	
Incision and drainage of pancreas. Died.	1	
Total................	101	
No history of previous gastrointestinal complaints.	12	
Previous gastrointestinal history. Attack.	55	
Previous gastrointestinal history, other than an attack.	15	
Surgical or pathologic findings proving the acute condition.	53	

TABLE II

Cases of Surgically Verified Acute Inflammation	No. of Cases	Notes
Acute cholecystitis or empyema. Cholecystostomy. Died.	5	1 had bronchopneumonia. 1 had severe cardiac disease.
Acute cholecystitis or empyema. Cholecystostomy. Lived.	13	1 had pancreatitis and fat necrosis. 2 had pericholecystitis.
Acute cholecystitis or empyema. Cholecystectomy. Died.	1	Had also pericholecystitis.
Acute cholecystitis or empyema. Cholecystectomy. Lived.	22	1 had a partial cholecystectomy done. 2 had pericholecystitis. 1 was chronic cholecystitis with old and new adhesions.
Ruptured gall-bladder. Cholecystectomy. Died.	1	
Empyema of gall-bladder with biliary sinus and pericholecystitis. Cholecystectomy. Lived.	1	
Acute cholecystitis, covered perforation, pericholecystitis. Cholecystectomy. Lived.	2	1 patient also had diabetes and pyelitis.
Chronic cholecystitis with necrosis and hemorrhage, pericholecystitis, adherent small intestinal loops. Cholecystectomy, enteroenterostomy, repair ventral hernia. Died.	1	
Subacute appendicitis, acute pancreatitis, acute cholecystitis, appendectomy, cholecystostomy. Died.	1	
Acute cholecystitis, cholelithiasis, choledochelithiasis, cholecystostomy, choledochostomy. Died.	1	
Empyema of gall-bladder, subacute hepatitis, pericholecystitis involving stomach, duodenum, and hepatic flexure, cholecystostomy. Died.	1	

TABLE II (Continued)

Cases of Surgically Verified Acute Inflammation	No. of Cases	Notes
Chronic cholecystitis, suppurative pericholecystitis, cholecystectomy. Lived.	1	
Empyema of gall-bladder, chronic appendicitis fibroid uterus, cholecystostomy, appendectomy, hysterectomy. Lived.	1	
Acute pancreatitis, acute hemorrhagic cholecystitis, cholecystectomy. Died.	1	
Acute cholecystitis in the presence of adenocarcinoma. Partial cholecystectomy. Lived.	1	
Total..............	53	

severely ill patient reaches the stage at which operation becomes imperative, he is usually a very poor surgical risk. Consequently, under these circumstances, the temptation is to perform cholecystostomy with or without drainage of a contiguous abscess. In such cases, the mortality is very high. (Table II.) It is reasonable to assume that the death rate would have been lower if expectant treatment had been abandoned at an earlier stage when the patients could have been operated upon with less risk. The patients would have been in better condition, the surgical procedure would have been simpler because the anatomic configurations would not have been so distorted.

If a patient with extensive inflammatory and structural changes in the biliary passages survives an operation, he still faces the possibility of a recurrence of symptoms and the necessity for a secondary operation. These points speak in favor of early operation.

Theoretically, early operation for acute cholecystitis is as desirable as it is for acute

appendicitis. But there is a practical difficulty: Many surgeons are capable of removing an acutely inflamed appendix; fewer are competent to remove an acutely inflamed gall-bladder. Clinically, it would seem that early operation would diminish the mortality from acute cholecystitis; but due to the lack of experience among many surgeons, the procedure may actually promote a higher death rate.

TABLE III

FOLLOW-UP DISTRIBUTION OF THE MEDICALLY TREATED CASES AND CASES SUBJECTED TO CHOLECYSTOSTOMY

Subsequent Course	Chole-cyst-ostomies	Medi-cally Treated	Total
Discharged from hospital.	17	20	37
Feeling well	7	6	13
Having complaints or objective signs	5	4	9
Had cholecystectomy done later .	2	2	4
Not traced	3	6	9
Having complaints but operation otherwise contraindicated	0	2	2

Reviewing the medically treated cases, we find twenty-three diagnosed as acute cholecystitis or empyema of the gall-bladder. (Three more medically treated cases in this series have no significance for this analysis; the diagnoses were: (1) acute inflammation of the liver and gall-bladder; (2) hepatic calculi and cholangitis; and (3) cholangiitis. In the first case there was serious doubt as to the existence of any real gall-bladder disease.) Of these twenty-three patients, two died; two refused operative consent; two left the hospital against the doctor's advice; and one was discharged unimproved only to be readmitted a few weeks later for cholecystostomy. In the remaining sixteen cases, the attending physician did not intend to apply any surgical treatment. One of these patients had a large palpable mass in the right upper quadrant with fever, rigidity and intense pain. The attending and the consulting physicians agreed that the process

was extensive but walled off, and that more could be gained by allowing the acute attack to subside than by prompt surgical interference. None of the remaining fifteen patients persistently exhibited symptoms suggestive of a very acute or serious condition. The mortality in this group was 8.7 per cent.

Fifty-three patients were operated upon and the verified diagnoses were acute cholecystitis and empyema of the gall-bladder. Some had been scheduled for medical treatment and were sent to operation only because the non-surgical therapy had failed. In such cases, the surgical mortality should really have been charged against medical treatment. In twelve of these fifty-three cases, serious complications (such as ruptured gall-bladder and other pathologic findings in addition to the cholecystitis) were found. Among the twelve were eight cholecystectomies with four deaths (50 per cent) and four cholecystostomies with three deaths (75 per cent). The number is small and the cases are not entirely comparable, but the fact remains that the cholecystectomies presented a better picture than the cholecystostomies. This is probably due to the tendency to submit the more serious cases to cholecystostomy.

The results in the remaining forty-one cases can be summarized in a phrase: twenty-three cholecystectomies with one death, and eighteen cholecystostomies with five deaths. The mortality is thus lower among cholecystectomies. The death rate is better than that reported from a study of a large number of chronic gall-bladder cases collected from the statistics of a mixed group of surgeons. In the latter review, the rate ranged from 4 to 9 per cent. Of course, our group is too small to be compared to this large series but it is extensive enough to refute the common statement that cholecystectomy in acute gall-bladder disease carries a very high mortality.

The immediate mortality, however, is not the only question. It is also important to know the ultimate condition of the

patients. The menace of persistent gall-bladder infection has often been stressed. Thus, it has been said that heart disorders may be caused or diabetes aggravated by latent gall-bladder disease; and that cholecystitis may set up a focus of infection. A discussion of this is beyond the scope of this paper. But we tried to determine whether cases treated medically or by surgical drainage subsequently developed complaints referable to the gall-bladder; and we also sought to discover the proportion of these patients requiring secondary cholecystectomy. Data on these points were secured from family doctors, from the operating surgeons and from personal interviews with the patients. The twenty-one medically treated cases and the seventeen instances of cholecystostomy represented thirty-seven patients, because one, first medically treated, reappears in the cholecystostomy group. Thirteen (seven cholecystostomies and six medically treated cases) felt well at the time of the follow-up investigation, from six months to four years after discharge from the hospital. Nine (five cholecystostomies and four medically treated) had definite complaints or objective symptoms (such as biliary fistula), while four patients (two in each group) had been rehospitalized for cholecystectomy. Two others (both medically treated) had definite complaints, but in these cases surgical treatment was contraindicated for other reasons. Nine (five cholecystostomies and four medically treated) could not be traced.

Roughly, half of the located patients who had not received primary cholecystectomy felt well. This might, perhaps, be construed, as evidence against cholecystectomy. The duration of the follow-up interval, however, was not very long and sooner or later some of these patients will probably develop gall-bladder symptoms again. A follow-up analysis of eighty-six cholecystostomies performed by Danzis[4] reveals that twenty-eight were subsequently subjected to secondary operations. He was obliged to do cholecystectomy from fourteen to twenty-two years after cholecystostomy in these cases. The incidence of reoperation was about 30 per cent.

SUMMARY

An analysis has been made of 101 cases, diagnosed as some kind of acute inflammation of the biliary passages. Of these, twenty-three were treated only medically and the diagnosis, though not verified, was probably empyema of the gall-bladder or acute cholecystitis. There were fifty-three surgically verified instances of acute cholecystitis or empyema of the gall-bladder. Twelve of these patients also had a serious complication. Mortality was greatest among the surgically treated complicated cases. Cholecystostomies showed a higher death rate than cholecystectomies, however, in these cases. The next lower death rate is shown by the uncomplicated cases treated by cholecystostomy, followed by the medically treated cases, while the lowest mortality was found among uncomplicated cases subjected to cholecystectomy.

CONCLUSIONS

Cholecystectomy in the hands of a competent surgeon is a desirable form of treatment for uncomplicated cases of acute gall-bladder disease. Its results compare favorably with cholecystostomy and with medical treatment.

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Sutures should be removed as soon as organic union has taken place. The longer they remain in the tissues after having served their purpose, the more likelihood there will be of a conspicuous scar.

From—"The Surgery of Injury and Plastic Repair" by Samuel Fomon (Williams & Wilkins).

AMMONIUM BICARBONATE SECRETED BY SURGICAL MAGGOTS STIMULATES HEALING IN PURULENT WOUNDS*

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UNUSUAL healing effects were obtained by Baer[1] some years ago when he placed living blowfly maggots directly into chronic non-healing wounds. His announcement of the success of this novel treatment attracted a good deal of interest, and during the next few years scores of articles appeared in the medical literature describing the remarkable results of maggot therapy.

The question then arose as to how blowfly maggots can promote healing. Baer apparently was the first to state his belief that these results are not due entirely to the scavenger action of the maggots in cleansing the wound. He conceived the idea that in addition "some biochemical exchange takes place between the maggot and the tissues of the patient." Later, in the Bureau of Entomology and Plant Quarantine, it was found that allantoin,[4,7] a metabolic product present in the maggot secretions, stimulates healing effects similar to those produced by the maggots. Further research in the Bureau showed that urea[5,6,7] also possesses this same characteristic.

Although this therapeutic property of allantoin and urea has since been confirmed and extensive use made of the two products by the medical profession, the possibility still remained that they are not the chief healing agents secreted by maggots. For one thing, they are produced only in minute quantities; and again, they are both regarded as inert substances physiologically.

One soon becomes aware of a metabolic product in maggot excretions which is both abundant and reactive. It is ammonia, and in the combined form in which it occurs in the excretions it is first ammonium carbonate, which is unstable, and then ammonium bicarbonate. In solution the latter has an alkalinity of pH 7.7, corresponding roughly with that of maggot excretions. Wounds in which maggots were used also tended to have this same alkalinity.

Uncombined ammonia is of course harmful to the tissues except in the minute amounts formed during metabolism; and any that is produced physiologically is believed to be removed by the liver. Maggots secrete comparatively large quantities of ammonium bicarbonate, along with other residue, directly into the wound, and this occurs without evidence of injury. Accordingly, some preliminary tests were made with this material to determine what effect the pure solution would have when applied to a wound.

The opportunity occurred in 1937 to treat a number of dogs and rabbits that had persistent discharging sores, chiefly on the back and legs. Two per cent solutions of ammonium carbonate and of bicarbonate in sterile water were made up and applied to the wounds in a series of comparative tests. As the use of gauze packs in these cases was out of the question, the wounds were thoroughly moistened several times daily with either of these solutions. The animals showed no discomfort and within two to three days the cleansing and healing effects formerly noted with allantoin and urea began to appear. No differences could be noted between the carbonate and the bicarbonate tests. The wounds, which had

remained open and running before treatment, healed with the formation of very little scar tissue and in some cases with a return of hair to the surface. The writer tried the bicarbonate later in the treatment of some minor injuries in his own case and also in some of his associates. When first applied it produced a slight stinging sensation which soon disappeared. In subsequent applications this smarting became less noticeable, and healing appeared to be normal.

The following year a number of physicians and surgeons* became interested in the therapeutic possibilities of these two ammonium compounds. The Bureau supplied concentrated sterile solutions and our coöperators diluted and used them in a few selected cases of discharging wounds of various types. The solutions were applied as 1 or 2 per cent concentrations on gauze packs saturated with the material and in some cases they were irrigated into the wounds.

The fact became evident as the treatments progressed that these two ammonium compounds are each able to promote healing in purulent and indolent wounds. Also no unfavorable effects were observed beyond the preliminary smarting in some cases. With this as a basis, treatments were then applied to a larger number of cases, including such purulent conditions as chronic osteomyelitis, diabetic and varicose ulcers, slow-healing abdominal wounds, abscesses, otitis media and in-

fected lacerations. Ammonium bicarbonate, being more stable and less alkaline, was used throughout these later tests.

An improvement in the condition of the wound similar to that produced by maggots usually began to take place after a few days' application of the treatment. The offensive odor decreased rapidly and the wounds became cleaner. Small areas of fresh granulations could be seen in the wounds and later a general development of this tissue usually occurred.

Following are some case histories supplied by our medical coöperators. They indicate the variety of purulent conditions which were treated, and the results appear to be typical of those usually obtained with this treatment.

CASE I. M. K., female, aged 16 years, had a chronic offensive discharge from the left ear. She had had two previous mastoidectomies and the ear had been treated with antiseptic powder insufflated frequently. On examination there was a slight discharge, stringy, clear and very offensive. Two per cent solution of ammonium bicarbonate was instilled into the ear and the patient complained of a stinging sensation. The solution was then diluted to 1 per cent and the ear cleansed daily for two weeks at this strength without discomfort. The concentration was increased thereafter until the patient was able to withstand a 4 per cent solution. After a period of eight weeks the discharge and odor disappeared, and after active treatment had been discontinued for six weeks no discharge or odor has been detected.

CASE II. C. H. K., female, aged 13 years, had had a swollen lower lip for six months. At the time of treatment the lip was considerably swollen and covered with crusts. Ammonium bicarbonate, 2 per cent strength, was applied to the lip on cotton packs for half an hour three times daily. In two days the swelling began to subside, the crusts had disappeared, and healing was well under way. Within ten days the lip was back to normal and healing was complete.

CASE III. S. W., male, colored, aged about 43 years, had chronic osteomyelitis of the left tibia. Multiple dirty sinuses developed, with malodorous drainage. Dressings with packs

saturated with 1 per cent ammonium bicarbonate were applied and kept constantly moist. These were changed daily. In ten days the odor disappeared and the discharge was markedly diminished. As treatments were continued the sinuses became smaller and healthy granulations appeared. The leg "felt better to the patient." In two months the wounds were all healed. X-ray films showed no appearance of osteomyelitis.

CASE IV. F. K., male, aged 40, had an old compound fracture of the right tibia with malunion and purulent anterior ulcer. This had been the condition for months. Three weeks' treatment with ammonium bicarbonate dressings reduced the ulcer by about 20 per cent and stopped drainage. The general appearance of the wound became healthier, and in five weeks healing was complete.

CASE V. W. D., female, aged 51 years, had a large chronic varicose ulcer on the lower left leg, of eighteen years' duration. On examination the ulcer was oozing a clear serum which produced a red rash on the surrounding skin. The entire lower limb was first cleansed with tincture of green soap and sterile water. Then applications of 2 per cent ammonium bicarbonate solution on saturated gauze packs were begun and changed daily. In about one week the ulcer cleaned up and developed a healthy base with good epithelial reaction around the margins. After six weeks' treatment the entire ulcer was healed and the patient was discharged and advised to have proper elastic stockings fitted.

CASE VI. A. R., a boy of 15, a chronic diabetic, received a deep laceration of Achilles' tendon. The wound was dressed and tetanus antitoxin was given. The patient went on a vacation but returned in ten days with a severely inflamed leg and a foul discharge from the wound. Temperature was 103 degrees. The wound was washed at once with 2 per cent solution of ammonium bicarbonate, and wet dressings with this material were begun. Both odor and pain decreased markedly and the wound dried up and was completely healed in seven days.

CASE VII. S. O. R., male, aged 36 years had severe lacerations of the right thumb and index finger, previously treated elsewhere with apparent healing. At the time of examination the wounds had broken down with excessive purulent discharge and considerable pain. Frequent applications of ammonium bicarbonate, 1 per cent solution, on cotton packs reduced suppuration and pain in two days, and in ten days the fingers were healed. Three months later they had remained healed.

CASE VIII. F. E. R., male, aged 52, had a "stitch abscess" following appendectomy and gall-bladder exploration. The patient was obese with low resistance of tissues. The wound was very slow in healing, twice breaking down with considerable drainage and odor, covering a period of five weeks. Finally frequent irrigations and daily wet pads of 1 per cent ammonium bicarbonate brought about cessation of discharge and appearance of firm granulations within ten days. In three weeks the incision was completely healed and has remained so for three months.

CASE IX. P. T. O., male, colored, aged 45 years, had had the eye ball removed following an injury to the right eye, but some time later the eye socket became infected. At the time of examination the socket had failed to heal and had discharged for fourteen months. The socket was then cleansed and packed daily with gauze saturated with 1 per cent ammonium bicarbonate. On complaint of pain, the concentration was reduced to one-half, when stinging ceased. In four days the odor and discharge were much reduced and two weeks later the socket was dry and healed.

CASE X. M. W., female, aged 64, had a chronic ulcer of the lower left leg due to arteriosclerosis. For one month the ulcer was treated with such drugs as thioglycerol, Ochsner's solution, methylene blue, and potassium permanganate without improvement. There was pain and high temperature, and a purulent fetid discharge was present. At that time treatment with a 1 per cent solution of ammonium bicarbonate on cotton packs was instituted. These packs were kept wet and renewed daily. In a few days the drainage began to subside and the edges of the ulcer cleared up somewhat. Small areas of pinkish granulation tissue started to grow in the center, and two months later the ulcer healed over and the patient was discharged.

CASE XI. P. B. T., male, aged 21 years, cut his right index finger on November 28, 1939 but had no medical attention given to it. One week later he had marked lymphangitis and cellulitis of the right arm with a temperature of 106 degrees. An incision over the original

injury released about 4 ounces of pus. The temperature returned to normal, but four days later it rose to 104 degrees with a huge cellulitis of right chest wall. An incision released clear serum. Ten days later another incision was made. Two weeks later the patient had a chill and a temperature elevation to 106 degrees. This was followed by a large empyema of the right side. Repeated incisions were made for three weeks and cultures continued to show streptococcus infection. A rib resection was then performed. Drainage and daily irrigations with Dakin's solution were given for four weeks with no improvement; then irrigations with neoprontosil, then with peroxide, then hexylresorcinol were used without any change. During this period from December, 1938 to April, 1939, sulfanilamide had been administered with no appreciable change noted. At this stage a 2 per cent ammonium bicarbonate solution was used to irrigate the chest cavity three times daily. On the second day of this treatment the pain in the lower chest disappeared and in four weeks the patient was out of bed with no signs of his ailment. He made an uneventful recovery and returned to his work.

CASE XII.   S. J. S., female, aged 72 years, had one or more varicose ulcers for twenty years. Upon her admittance to the hospital the ulcers had a fetid odor and were badly necrosing. Also the leg was very swollen. The ulcers were thoroughly washed with 1 per cent ammonium bicarbonate, and gauze packs saturated with this material were applied and changed daily. Within two days the odor had disappeared and the necrosed areas were much cleaner. In two weeks the wounds became exceedingly clean and full of fresh granulations and blood vessels. At this time the bases of the ulcers were covered with autogenous pinch grafts, forty-seven in all. Two weeks later, by actual count, forty of these grafts were found to have grown beautifully and the ulcers were practically cured. Usually not more than 50 per cent of pinch grafts take and they have to be repeated from one to six times. The age of the patient and the fact that she had had the ulcers for twenty years must be considered.

Ammonium bicarbonate is readily available commercially and is made on a large scale for various purposes. It also has the advantages of being cheap and easily prepared for use. One-pound bottles can be purchased for about forty cents. At that price a liter of 1 per cent solution costs one cent. A stock solution is made by adding 10 Gm. of the bicarbonate to each liter of cold water and stirring occasionally until dissolved. It goes into solution rapidly. If the containers are tightly corked, both the crystals and the solution will keep well.

A fairly wide range in the strength of the solution has been used with success. This has varied from $\frac{1}{2}$ of 1 per cent to as high as 4 per cent concentration. A growing tolerance to the bicarbonate with repeated applications has been reported in several instances. In some cases the first few treatment have been made with $\frac{1}{2}$ of 1 per cent concentration and then raised to about 2 per cent without discomfort and with especially favorable results in promoting healing. In some reports it is especially stated that the patients were free from discomfort during treatment. Surfaces around the wound not needing treatment are sometimes given a light application of vaseline.

Wounds tended to become alkaline when they were implanted with maggots, and it was believed that such wounds healed more rapidly than those inclined to be acid. Messer and McClellan,[3] in their early study of the hydrogen ion concentration of wounds treated with maggots, also noted: "In all cases the fluctuations in pH corresponded with the clinical condition of the wound; the progress of healing being accompanied by a consistent increase in alkalinity while failure to progress toward healing . . . was followed by a marked fluctuation toward acidity." The application of ammonium bicarbonate brings about an alkalinity in the wound similar to that produced by maggots.

A catalyst with a background of unusual chemical interest, namely, the enzyme urease, is apparently the agent responsible for the production of ammonium bicarbonate in maggot secretions. This enzyme, which has been known since 1860, was recently found by Robinson and Baker[8] to occur abundantly in the tissues and excre-

tions of surgical maggots. Urease is specific for the metabolic waste product urea, and it cannot react upon even closely related substances. It is a heavy protein molecule with a molecular weight of 483,000, according to Sumner et al.,[9] while urea is of small size with a molecular weight of 60. Urease hydrolyzes urea and produces ammonium carbonate by means of two successive reactions (Van Slyke and Cullen[10]). A molecule of urease first combines with one of urea. Then it immediately disrupts the urea molecule. From this reaction the enzyme emerges free, and if another molecule of urea is available urease at once reacts upon it without any measurable loss of time. The carbonate, as stated earlier, is unstable and with a loss of one molecule of ammonia it becomes ammonium bicarbonate. Brown[2] found the bicarbonate in maggot excretions but his technique did not show the presence of the enzyme urease.

Urease, urea, and the resulting ammonium compounds comprise a system in living tissues which appears to be of especial interest and significance. A consideration of that system, as it bears upon the growth of tissue in healing wounds, has been part of the investigation here reported, and it will be discussed in a forthcoming article in this Journal.

SUMMARY

In a further analysis of the secretions of surgical maggots, ammonium bicarbonate has been found present in comparatively large quantities. With the aid of a number of physicians and surgeons this product has been tested clinically and found to have marked healing properties similar to the other two maggot products, allantoin and urea. Ammonium bicarbonate is inexpensive and is easily prepared for use in solution form. It was applied to purulent wounds as a 1 or 2 per cent sterile solution on gauze packs. The occurrence of this compound in maggot secretions is associated with the presence of the enzyme urease in maggot tissues. The blowfly *Phormia regina* was the species used.

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CASE REPORTS

TERATOMA OF THE SPERMATIC CORD; CASE REPORT WITH A CONSIDERATION OF THE PROLAN TEST*

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NEOPLASTIC tumors of the spermatic cord are rare; there are about 200 recorded cases. Of the malignant tumors, some form of sarcoma is the most common, while the teratoma is seldom encountered, although it is difficult to say how many actually have been reported. MacKenzie groups them as "embryonal tumors," including in this classification the dermoid teratoma, myxosarcoma, fibromyosarcoma, etc. Thompson in his review of tumors of the spermatic cord mentions no other cases of teratoma than the one reported by Rubaschow in 1926. Hinman and Gibson state that "analysis of cases reported as mixed tumors and teratomas of the cord reveals a confusion of terminology and lack of detail which in many cases casts doubt on their authenticity." With this we concur.

We prefer to reserve the term teratoma for those tumors which contain tissues derived from any or all three germinal layers, with at least two present. In contrast to teratoid tumors, the various types of sarcoma, such as the myxoliposarcoma for example, represent true connective tissue tumors, derived from one germinal layer, the mesoderm. A tumor resembling a sarcoma histologically may result from the one-sided development of the mesothelial elements of a teratoma, but careful search of the specimen should demonstrate the products of one or both of the other layers.

In contrast to the relative rarity of teratomas of the spermatic cord, teratomas of the testicle are common, and a great deal of work has been done in the biologic assay of the hormones associated with this group of tumors. Zondek in 1930 was the first to report the results of tests for prolan A in the urine from a patient with a teratoma of the testicle. Ferguson, Cutler, and Owen have published the results of further studies in this field.

The case of teratoma of the spermatic cord which we are reporting is of special interest not only because of its rarity, but because of the application of the qualitative test for prolan on several occasions. It seems reasonable to assume that there is no more important difference biologically than there is histologically between a teratoma of the testicle and one of the spermatic cord, and that therefore the application of prolan tests to tumors of the spermatic cord should be of equal diagnostic and prognostic value. For those who are not familiar with these tests, it may be of interest to briefly state the current views regarding them.

Two hormones are concerned: prolan A (F.S.H. or follicle-stimulating hormone) and prolan B (A.P.L. or anterior-pituitary-

like-hormone). Normally prolan A is found in the urine at various times during the menstrual cycle. It may also be found in the urine from the normal male, but in such small amounts (i.e., below 100 mouse units per liter) as to be demonstrable only by the most sensitive of the biologic tests. When, as a result of orchidectomy, ovariectomy, menopause, irradiation of the testicles, cerebral tumors, etc., the hormonal balance of the body is disturbed, markedly increased amounts of prolan A may be found in the urine due to hyperactivity of the anterior pituitary gland. Prolan B, on the other hand, is not found in the normal individual. It is present in large amounts in the urine of pregnant women, especially during the first three or four months of pregnancy. It is believed to be elaborated by the placenta, probably by the cells of the chorion. It is the hormone concerned in positive prolan tests of patients with teratoid type of tumors, when they contain cells like chorionic epithelium. Urine from cases of chorionepithelioma contains, as might be expected, the largest amounts of prolan B; up to 100,000 and more mouse units per liter have been found. In brief, it may be said that a positive prolan test in a patient with a testicular or spermatic cord tumor is indicative of an immature teratoma. The reappearance of a positive test following a negative one in a treated case may be due to recurrent disease (either local or metastatic) or to hormonal imbalance as a result of the orchidectomy or irradiation of the genital area. Cases with recurrent disease generally give larger amounts of prolan or increasing amounts on successive tests, as contrasted with relatively low or stationary levels in patients with mere hormonal imbalance.

An American laborer, aged 72, was admitted June 27, 1935 with a complaint of swelling of the right side of the scrotum. Six months before admission he noticed enlargement of the testicle upon that side and there followed a rapid and progressive increase in size. Some relief was obtained from the wearing of a suspensory, but when the mass became quite large, hard, and tender, a physician was consulted and hospitalization was advised. The patient stated that he had lost 16 pounds in weight. He reported also that he had never had a testicle upon the left side. He was married and had had two children. There was a record of Neisserian infection many years before. Otherwise the history was unimportant.

Examination found a well-nourished man of 72, apparently in good general condition. The right side of the scrotum was markedly enlarged and contained a freely movable, nontender, coarsely lobular mass. It did not appear to be adherent to the scrotal skin. The lower pole of the tumor was soft and fluctuant and capable of transillumination. The upper pole was sharply defined and above it, a somewhat thickened cord could be felt at the external ring. There were no enlarged glands in the groins except for a few small nodes in the right inguinal region. The left testicle was absent. No abnormality was found on abdominal and rectal examination. X-ray films of the chest revealed no evidence of metastatic disease. The blood Hinton test was negative, and the test for prolan in the urine was positive.

Although the advanced age of the patient rendered the clinical picture unusual, we believed that we were dealing with a rapidly growing tumor of the testicle, probably malignant. In accordance with our routine procedure, therefore, in such cases, high-voltage deep x-ray therapy was given as the first step in treatment. The anterior and posterior aspects of the scrotum received 1200 R units each. No demonstrable regression of the tumor was noted, and one month after the completion of the x-ray series, a right orchidectomy was performed under spinal anesthesia.

A small oblique incision was made in the groin on the right side above and parallel with Poupart's ligament, avoiding the region of the tumor. A few small inguinal lymph nodes were encountered and removed; one of them, examined by frozen section, did not show malignant disease. The spermatic cord was freed by blunt dissection after dividing the external oblique fascia in the line of its fibers, and the cremaster muscle. The cord was carefully freed up to the internal ring, and palpation through the ring along the external iliac artery detected no nodes in this region as far as the finger could reach. The cord was then doubly ligated at the internal ring with No. 1 chromic catgut, and

divided with high-frequency current distal to the ties. At no time was a clamp placed upon the cord. The distal stump of the cord and the

FIG. 1. Photomicrograph of tumor showing undifferentiated cells and fibroblastic portions. (× 300.)

scrotal tumor were then carefully enucleated, chiefly by blunt dissection, after enlarging the incision downwards onto the anterior wall of the scrotum. The wound was closed in layers and the patient left the operating room in good condition.

We wish to emphasize the importance of the careful and gentle division of the spermatic cord at the level of the internal ring, before the scrotal contents are disturbed in any way, in all cases of orchidectomy for testicular neoplasm.

Pathologic Report. About 4.5 cm down from the upper cut end of the cord was a well encapsulated, oval tumor, measuring 11 × 8 × 5.5 cm. This was fairly firm and somewhat tense. The vas deferens swung over the surface of the tumor to the testis without being involved in the tumor though the various sheaths of the cord appeared to be incorporated in the capsule.

The testis and epididymis were soft and definitely separated from the tumor. The

testis was 3.5 cm. in diameter, composed of grayish-yellow, semi-translucent tissue with yellowish-white strands and scattered brownish

FIG. 2. Photomicrograph showing variation in structure and tumor giant cells. (× 200.)

pigmentation. The tunica vaginalis was thickened and there was a hydrocele about 4 cm. in diameter. The epididymis was negative.

On section the tumor was soft, grayish-brown to yellowish-white, with fibrous strands radiating from a rather fibrous center toward the periphery.

A mass of fibrofatty tissue 3 cm. in diameter containing several small resilient lymph nodes was present; this measured up to 0.6 cm. in diameter. On section the nodes were somewhat fibrotic and dark yellowish-gray.

The tumor was loose textured, with numerous large, ill-defined cells, some of which were multinucleate. Throughout, the general groundwork of the tumor was made up of immature spindle-shaped cells varying markedly in size and having a variable amount of very loose intercellular fibrils adjacent to them. In some places fairly dense strands of collagen with well-formed fibroblasts were present. Irregularly scattered among these spindle cells were

large, rounded to elongate, or even stellate, cells having either a single large irregular nucleus or multiple nuclei. The cytoplasm contained numerous small granules or, at times, definite myofibrils with cross striations. Here and there a few of the larger of these cells were vacuolated, with a definite spider web arrangement of the myofibrils.

A moderate number of vacuolated cells resembling immature fat cells were present and rare adult fat cells occurred. In some regions close-packed spindle cells with elongate, blunt-ended nuclei were present. Intercellular substance did not occur between these cells, which apparently were smooth muscle cells. Here and there were some foci of small ill-defined, close-packed, polyhedral cells. Mitoses were very rare in all types of cells. Rare foci of lymphocytes were noted, chiefly in relation to vessels.

The lymph nodes were slightly fibrotic, the epididymis negative. The testis tubules were atrophied. Large masses of compact polygonal cells with round nuclei contained scattered chromatin. Some yellow-brown pigment granules were present. The wall of the hydrocele was composed of fibromuscular tissue containing smooth muscle. Slight lymphocytic infiltration was observed.

Pathologic diagnosis was teratoma of spermatic cord, predominantly mesenchymal; hyperplasia of testicular interstitial cells; negative nodes.

The surgical convalescence was quite uneventful. Two weeks after the operation no prolan was found in the urine. Three weeks after orchidectomy deep x-ray therapy was resumed and 1200 R units were given to the anterior and posterior aspects of the pelvis. Somewhat less treatment was given than had been planned because of the onset of diarrhea. The patient was discharged to the out-patient clinic, September 22, 1935.

He was seen one month later when he reported that he had gained 11 pounds and was back at work. He failed to return to the Clinic until April, 1936, at which time it was found that the prolan test was again positive. The general condition was good and the examination was satisfactory, except that in the right upper quadrant of the abdomen there was a vague suggestion of a mass. The abdomen was quite full in this case and difficult to palpate accurately. Readmission to the hospital was advised because of the prolan test, and because

of the possibility of recurrent disease in retroperitoneal glands.

Deep x-ray therapy was given in June, 1936.
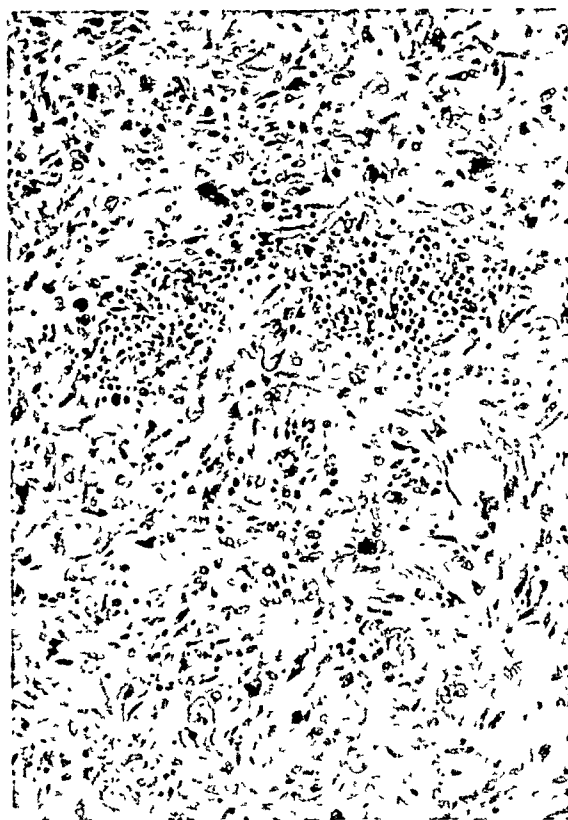
FIG. 3. Myxomatous regions and tumor giant cells. (× 300.)

The right and left sides of the abdomen were treated, anteriorly and posteriorly, and 1200 R units were given through each portal. X-ray films of the chest were negative. The patient left the hospital June 24, 1936. The next prolan report on July 27 was negative.

It should be emphasized that in our routine management of cases of malignant disease of the testicle, usually in much younger individuals and with much more definite pathologic pictures, we consider the fields for deep therapy to include the chest and neck as well as the abdomen and pelvis. A negative x-ray film does not preclude the possibility of active disease beginning in the lungs and mediastinum. In the case under discussion, the advanced age of the patient and the unusual character of the tumor, together with the lack of any definite evidence of its recurrence led to a modified program of treatment.

The next out-patient visit was September 23, 1936. The patient reported that he had been quite well and he had no significant complaints.

At follow-up visits two and five months later there was no change in his condition, and no evidence of recurrent disease. The prolan test carried out on December 5, 1936 was positive.

He was readmitted to the hospital on March 2, 1937. He had gained 20 pounds during the previous six months. He was thoroughly studied at this time. Examination of the upper gastrointestinal tract by fluoroscopy showed a displacement of the lower esophagus which caused some delay to the passage of the barium, but produced no deformity in outline or in the pattern of the mucous membrane. Extrinsic pressure by a tumor mass lying outside the esophagus was considered. However, a film of the chest showed the lung fields to be clear. It was finally decided to irradiate this area and the patient was given 1200 R units each to the lower mid-chest and right upper abdomen. Clinical examination revealed no evidence of local or metastatic disease except the questionable area above. A barium enema was negative for disease in the colon. X-rays of the pelvis and lumbar spine showed only arthritic changes. A Graham test showed no filling of the gall-bladder with dye and was interpreted as indicating an abnormal gall-bladder. This, perhaps, was the explanation of the increased resistance to palpation previously noted in the right upper quadrant and the suggestion of a palpable mass in that region.

The patient was discharged twelve days after admission. A week later, March 18, 1937, the prolan test was reported as slightly positive. Two months later he was readmitted to the hospital for a brief stay, and reëxamination of the esophagus at this time showed the same deviation to the left, and no change in the lung fields. No treatment was given. The prolan test, repeated on June 1, 1937, was positive.

The patient did not report to the clinic again until March 9, 1938. At that time he had no complaints of any kind and weighed 5 pounds more than when he entered the hospital. Examination showed no evidence of recurrent or metastatic disease. His urine was tested for prolan and was negative.

Since quantitative determinations were not done we cannot state definitely that the various positive prolan reports following operation were not due to recurrent disease. However, at his last visit to the clinic, over two and one-half years after his operation, no clinical or biologic evidence of disease could be found, and we believe we are justified in assuming that the previous positive tests were due to hormonal imbalance caused by hyperactivity of the pituitary following castration.

SUMMARY

1. The report of a case with a true teratoma of the spermatic cord, clinically free of disease for over two and one-half years is presented.

2. The significance and value of the determination of urinary prolan in spermatic cord tumors is briefly discussed.

The patient was last seen in the Urological Clinic on August 2, 1939. He had no urinary complaints. His only difficulty was edema of the lower extremities which could be explained on a cardiorenal basis. There was no clinical evidence of metastases.

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TORSION OF THE SPERMATIC CORD

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THE importance of an early accurate diagnosis and the prompt institution of surgical treatment, and the increasing frequency of recognition of torsion of the spermatic cord, warrants a brief view of the salient features and the report of a characteristic case.

A review of the recent literature of torsion of the spermatic cord discloses a considerable discrepancy between the reported and the actual incidence of the condition. In a recent painstaking review of the literature, Abeshouse[1] states, that up to January 1, 1936, there were 347 cases reported; consequently, the condition is relatively rare. In 156 cases he found only twenty-four in infants less than 1 year of age.

Torsion of the spermatic cord is caused by a sudden axial rotation or twisting of the spermatic cord. The blood vessels of the cord are constricted, resulting in an acute circulatory embarrassment in the testis and adnexa, producing local, and occasionally general symptoms. The condition is described as torsion, volvulus, strangulation, gangrene or infarction of the testicle. The term torsion of the spermatic cord is more descriptive of the pathologic process involved.

Torsion may occur at any age, but is most frequently observed in the first two decades of life. It is found as often on the left as on the right side.

The etiology has not been definitely determined. It is generally agreed to be due to some defect in the anchoring of the testicle and epididymis to its bed. The two essential factors involved, upon which the pathology is dependent, are, (1) hypermobility of the testicle, due to a congenital or acquired anatomic abnormality of the testis or its adnexa—this is considered the chief predisposing factor; and (2) an internal or external force resulting in strong contraction of the cremasteric muscle, which is considered the chief exciting factor.

The mechanism of torsion varies with the anatomic structures involved, whether intravaginal and extravaginal. In the intravaginal, the type commonly observed, the twisting of the cord lies entirely within or beneath the tunica vaginalis. The testicle and the epididymis, and not the tunica vaginalis, are involved in the process. In the relatively rare extravaginal type which usually occurs in an undescended or intra-abdominal testis, the twist lies outside the tunica vaginalis and involves this structure as well as the testicle and epididymis. The number and direction of the twists in the cord vary. The degree of torsion may be 90 to 360 degrees. The direction of the twist is usually clockwise on the right and counterclockwise on the left.

The pathology found is the result of obstruction or occlusion of the spermatic veins by the twist in the cord. Early, the testis and epididymis are congested and hemorrhagic; later there is a characteristic hemorrhagic infarction and aseptic gangrene. Atrophy or suppuration may ensue, and a complete disappearance of the testis and epididymis often follows.

The symptomatology is not pathognomonic but is very suggestive, and is directly attributable to the acute circulatory disturbance resulting from the torsion. Usually the following symptoms are found:

1. The onset is sudden with severe pain in the testicle, usually accompanied by nausea, vomiting and mild shock.

2. Swelling of the testicle and epididymis with redness and edema of the skin either in the scrotum or inguinal canal is noted.

3. The twist in the cord may be palpated.

4. Pain is increased on elevation of the testicle (Prehn's sign).

5. There is rotation of the epididymis to a lateral or anterior position.

6. The scrotal contents are retracted upwards.

7. Mild general or constitutional symptoms occur.

8. The leg on the affected side is held in flexion.

9. There is no impulse on cough, or enlargement in size upon crying of the irreducible tumor.

In torsion of the incompletely descended testicle, the accompanying hernial sac or peritoneum in the region of the ectopic testis may become irritated or inflamed and tense, giving rise to all the symptoms of early strangulated hernia. Palpation of the involved structures offers no aid in diagnosing such cases. In addition to the acute type above described, a subacute and a chronic or recurring type is also occasionally seen.

The differential diagnosis is particularly important because of the obvious difference indicated in treatment. Torsion must be differentiated from:

1. Strangulated inguinal hernia which usually occurs at a later period of life. Its course is of rapidly increasing severity, the general systemic and intestinal symptoms are usually more severe than in torsion, although the local symptoms may be identical. The two conditions may coexist.

2. Acute orchitis, where the pain is usually not so severe, the onset gradual, and the swelling more extensive.

3. Acute epididymitis, usually gradual in onset. Other evidences of a local infectious process may be found in the urine, urethra, prostate, and vas deferens, which are absent in torsion.

4. In acute hydrocele or hematocele the pain is not so severe as in torsion, there is no upward retraction of the scrotal mass,

and transillumination is possible if the fluid is not too bloody.

5. Tumors of · the spermatic cord or inguinal glands, because of slow onset, offer no diagnostic difficulties.

6. Suppurative inguinal adenitis is usually gradual in onset, the pain is not so severe, and careful search reveals a local infectious process responsible for the adenitis.

The prognosis is invariably good. Not a single fatal case has been reported.

Treatment must be promptly instituted and depends upon proper recognition of the true nature of the disease. Gangrene or atrophy occurs in about 85 per cent of cases not relieved by immediate operation. Prophylaxis aims to correct the predisposing causes of torsion, namely, undescended testis and hypermobility of the testis. It consists of the giving of anterior pituitary-like hormone in cases of undescended tests. If torsion actually has occurred, detorsion must be accomplished. Non-operative detorsion of the unexposed testicle is unsatisfactory and does not prevent recurrence.

Operative treatment is either (a) detorsion and orchidopexy, or (b) orchidectomy. The former represents the ideal form of treatment and offers the greatest chance for preserving the testis; it consists of the transposition of the testicle into the scrotum if undescended and of fixation or anchoring of the testicle posteriorly to prevent later recurrence of torsion. Orchidectomy is the operation of necessity and is indicated in those cases presenting gangrene of the testicle and epididymis or a persistent circulatory disturbance.

CASE REPORT

R. S., age 4 months, was admitted to the Children's Hospital November 11, 1937. He had been seen in the office one hour earlier upon the complaint of his mother that he was irritable, held his left thigh in a flexed position, and had a hard lump in the left groin. These symptoms had appeared about eighteen hours previously along with loss of appetite. There had been no vomiting, the morning stool was normal, and no urinary disturbance was pres-

ent. Pain was apparently severe, as the infant cried almost continuously.

Examination showed a rectal temperature of 99.5, pulse 120, respiration 24. The left thigh was held in a flexed position, and attempts to extend it resulted in an increase of pain. There was spasticity of the musculature of the entire left lower quadrant. In the left inguinal canal, at the level of and just below the internal ring, a hard irreducible mass was felt, very firmly adherent to the underlying structures, which evidenced no impulse on cough, and became no larger with crying of the infant; it was very tender to touch. The skin overlying the mass was congested and edematous; the mass measured about 1½ inches long by 1 inch wide. The testicle could not be definitely palpated in the left scrotum, but was palpated in the right. The urine was negative. The leucocyte count was 19,600 with 68 per cent polymorphonuclears.

Preoperative diagnosis of (1) strangulated left inguinal hernia or (2) torsion of left spermatic cord was made. Operation was performed within two hours from the time the patient was first seen and within twenty hours of the onset of symptoms.

Under ether anesthesia an incision was made over the mass in the left inguinal region, cutting through a very edematous skin and opening the left inguinal canal. A blue domed mass was uncovered, tightly adherent, about 1½ by 1 inch in diameter; the cord was not found to extend below this mass. The thick walled sac surrounding the mass was completely freed and gently opened, considerable dark bloody serum was evacuated, and a bloody gangrenous mass exposed. This was found to consist of the testicle, which looked like an organized blood clot, attached to its cord, which had two complete twists in it in a clockwise direction. As the testicle was in a hopeless condition, the cord structures were isolated, ligated separately, and the testicle and involved portion of the cord resected. The sac communicated with the general peritoneal cavity but contained no bowel; the sac was ligated and transfixed and the usual Halstead closure made. The patient made an uneventful recovery and was discharged from the hospital nine days later.

Pathologic Report. The testicle and epididymis were swollen to about twice normal size, were softer than normal, bluish black in color and the consistency of a blood clot.

There were many extensive confluent areas of extravasated blood filling the interstitial spaces. The blood vessels revealed considerable enlargement of the lumina which were crowded with red blood cells. There was also considerable extravasation of blood in the ducts of the epididymis. The testicle showed very extensive degenerative changes and there was almost a complete disappearance of the nuclear structure of the spermatogenic cells. There were also disseminated necrotic areas throughout the entire interstitium. The capsule appeared moderately thickened and a slight amount of fibrin deposit was noted on the tunica vaginalis testis.

SUMMARY

A brief review of the interesting factors in the anatomy, diagnosis and treatment of torsion of the cord has been given and a case report added.

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MUCOCELE OF THE APPENDIX*

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CYSTIC dilatation of the appendix was first described by Virchow[1] in 1863, and was given the name "mucocele" by Feré[2] in 1877. To date approximately 400 cases have been described. Dodge[3] collected 142 cases up to 1916, and Weaver[4] in 1928 was able to add 26 additional cases, including two of his own. Castle[5] collected post-mortem statistics, finding twenty-nine mucoceles of the appendix in 13,158 post-mortem examinations, an incidence of approximately 0.2 per cent. Dannreuther[6] reported eight cases in a series of 8,457 appendectomies at the New York Post-Graduate Hospital. Mayo and Fauster[7] record seventy-six cases out of a total of 31,200 appendectomies. Jirka and Scuderi[8] found an incidence of 0.23 per cent in surgically removed appendices at the Cook County Hospital, as compared to an incidence of only 0.043 per cent in an almost equal number of autopsy specimens, indicating that symptoms are severe enough to lead to operation in the majority of cases. The greatest incidence was six cases in 400 autopsies (1.5 per cent) reported by Ribbert.[9]

The etiologic factor as given by Deaver,[10] Kelly,[11] and others is the local obliteration of the lumen of the appendix with cystic dilatation taking place distal to the site of the obstruction. Phemister[12] states that there is a direct relationship between the development of a mucocele and the normal involution of the appendix. This belief is based on the fact that most cases occur after the age of 35 years when retrogressive changes and obliteration of the appendiceal lumen take place. However, he states that the appendiceal lumen need not be completely obliterated. The pseudomucin escapes into the cecum, a condition demonstrated roentgenologically by Vorhaus[13] and Lifvendahl.[14] A patent lumen was demonstrated in my second case.

Mucocele involving both an ovary and the appendix was described first by Bailey[15] and later by Ries[16] and others. Eden[17] and Rathe[18] have recorded cases in which pseudomyxoma peritonei secondary to ruptured ovarian cysts was found at the first operation and a cystic appendix was removed at a later date. These occurrences of a mucocele of the ovary and the appendix in the same patient have been frequent enough to cause one to speculate as to the possibility of some other etiologic factor. Further argument in favor of other than obstruction as an etiologic factor is the occurrence of mucoceles involving a portion only of the circumference of the appendiceal wall.

While most cases occur after the fourth decade, they are by no means limited to this age. Norment[19] found the average age of patients with mucocele of the appendix in a large series to be 41 years. His youngest patient was 4 years old and the oldest 65. He found the ratio of males to females to be 2 to 1.

The pathologic features are characteristic. The tumor varies in size from a small cyst about 1 cm. in diameter to the size of a man's head, as reported by Neuman.[20] The shape varies with the size and the extent of the appendix involved. It may be oval, round, cylindrical, curved, nodular, clubbed or sausage-shaped. The content is usually thick and gelatinous, but may be watery, and a few cases have been reported in which the content resembled fish eggs.[21–22] Clinical tests show it to be pseudomucin. The entire appendix may be involved and the base may project into the cecum so as to make removal difficult.

The symptoms are not diagnostic and may be entirely absent. About half of the patients have vague distress in the right

FIG. 1. Drawing of specimen of Case 1. Insert shows invagination of base of appendix into cecum.

lower abdominal quadrant. In one-fifth of the cases reported by Mayo and Fauster[7] a mass was palpable. This palpable mass usually leads to the diagnosis of ovarian cyst or a lesion of the bowel. Roentgenologic evidence is not diagnostic. Only two cases have been diagnosed preoperatively.[13–23]

The most serious complication is rupture into the peritoneal cavity with development of pseudomyxoma peritonei. Werth[24] first described this condition arising from a mucocele of the ovary, and Fraenkel[25] in 1901 was the first to show pseudomyxoma peritonei arising in the male from the rupture of a mucocele of the appendix into the peritoneal cavity. Acute inflammation of the appendix superimposed on a mucocele may occur as in a case reported by Christopher,[26] and as in my second case and in a few others.

CASE REPORTS

CASE I.   The patient, a white male 65 years of age, had never complained of any discomfort referable to his abdomen. Six weeks previous to being seen by me he had a right inguinal hernia repaired. A firm white mass was palpated in
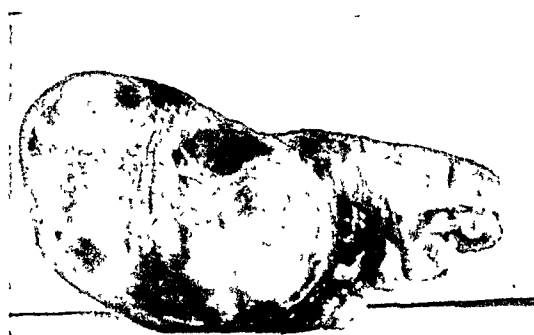
FIG. 2. Photograph of appendix in Case II.

the region of the cecum, but could not be delivered. A diagnosis of carcinoma of the cecum was made at the time of the operation, and after recovery from the herniotomy, he was referred to me for a bowel resection.

The patient was well developed, well nourished, and had not lost any weight. No gross or microscopic blood was present in the stools when on a meat-free diet. A mass was palpable in the region of the cecum. On x-ray examination a filling defect of the cecum was noted, suggesting that the diagnosis of carcinoma of the cecum was correct.

At operation a mucocele involving the entire appendix was found. The appendix was erect, firm and its base projected into the cecum so that about 25 to 30 per cent of its length was surrounded by the wall of the cecum. There was no evidence of pseudomyxoma peritonei.

The entire cyst was removed by enucleating it from the cecum. Since closure of the defect in the cecum would have compromised the ileocecal valve, a large Pezzer catheter was placed in the cecum and brought to the outside of the abdomen through a stab wound.

The patient made an uneventful recovery, and the fistulous tract to the cecum closed spontaneously after removal of the catheter.

Pathology. The appendix was 10 by 5 cm. (after fixation), sausage-shaped, and tensely distended. The distal two-thirds was covered with smooth and glistening peritoneum, while in the proximal third the muscularis was exposed. The color was white with a faint tinge of pink. A small amount of thick mucus-like material escaped from the cut end.

Since the appendix was to be preserved as a museum specimen, no sections were taken for microscopic study.

CASE II. A male 60 years of age had felt "uncomfortable in the lower abdomen" for a week previous to consulting his physician. On the day of admission to the hospital the patient had been nauseated and had vomited once. He complained of a constant pain in the lower abdomen to the right of the midline. Anorexia was present. A history of auricular fibrillations was given.

Physical examination revealed a temperature of 100.6 degrees, pulse 96. The blood pressure was 122/88 m.m. of mercury. The patient obese, florid-faced. A fairly constant extrasystole was present. The abdomen was obese but not distended. Tenderness was noted on pressure in the right lower quadrant of the abdomen, but not in other parts of the abdomen. Increased muscle tone was present in the right lower quadrant of the abdomen. No mass was palpable through the abdominal wall. Pressure in the right flank did not produce pain. On digital rectal examination a tender mass was palpable high in the pelvis on the right side. On auscultation of the abdomen peristaltic sounds were heard throughout, but were diminished in frequency over the right lower quadrant. No obstructive borborygmi were heard. The leucocyte count was 14,550. The urine was normal except for a trace of bile which persisted throughout the patient's stay in the hospital.

A preoperative diagnosis of acute pelvic appendicitis was made. At operation free fluid was found in the peritoneal cavity. The terminal ileum and cecum were hyperemic and bound down by fibrinous adhesions about a large firm mass extending over the brim of the pelvis. This mass was a large bulbous appendix, gangrenous in part, while other portions were either semi-transparent or white like scar tissue. The wall of the proximal half of the appendix was hyperemic and greatly thickened and indurated, the induration involving the cecum at the base of the appendix and causing difficulty in removal. The cecum was closed after removal of the appendix without obstructing the ileocecal valve. There was no evidence of pseudomyxoma peritonei. The abdominal wall was closed without drainage.

The postoperative diagnosis was infected mucocele of the appendix.

The convalescence was uneventful save for an auricular fibrillation which was controlled by quinidine and a singultus which was controlled by barbiturates.

Pathologic Examination. The appendix measured 10 by 4.5 by 2.5 cm. The distal end was bulbous and enlarged to a diameter of 4.5 cm., and filled with bloody exudate and mucus. The wall of this portion was mostly thin with thickened white areas. After removing a section for microscopic examination, a gelatinous-like mucus was found adherent to the wall of the bulbous portion. The lumen of the appendix was patent.

Microscopic examination revealed moderate infiltration of the walls by both neutrophiles and round cells. A marked fibrosis of the mucosa and submucosa indicated a process of long standing. Both the lymphoid tissue and the mucosa were almost completely replaced by fibrous tissue. Instead of normal appearing glands, the structures had the form of papillary projections from the inner wall. The epithelium about the tips of these projections was mostly of the mucous type.

COMMENT

Symptoms of mucocele are frequently severe enough to cause removal of the appendix, although a preoperative diagnosis is a rarity. The theory that the lumen of the appendix must be blocked proximal to the mucocele and that the content must be sterile is not always correct, as illustrated by the second case reported in this paper.

The concomitant presence of a mucocele of the appendix and of an ovary suggests the possibility of a developmental defect or congenital precursor. Rupture of a mucocele of the appendix or ovary may give rise to pseudomyxoma peritonei.

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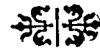
MESENTERIC THROMBOSIS—OPERATION—RECOVERY*

REPORT OF TWO CASES

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THROMBOSIS of the superior mesenteric vessels is an infrequent cause of intestinal obstruction. McIver, in an analysis of 335 cases of intestinal obstruction, found that only 3 per cent of the obstructions were due to occlusion of mesenteric vessels. Donaldson and Stout have contributed to the pathology of this affection and by their careful clinical observations and animal experimentations have differentiated between arterial and venous mesenteric thrombosis. They point out that with venous thrombosis the muscular structure of the intestinal wall remains viable for a considerable period of time, whereas in arterial thrombosis, gangrene of the intestinal wall comes on immediately.

Both conditions need surgical intervention but it is more urgently required in the arterial type. The mortality without surgery in thrombosis of the superior mesenteric artery is 100 per cent. The occasional reports in the literature of recovery without operation must have been examples of thrombosis of the superior mesenteric vein. The mortality rate even with operation is extremely high. It is estimated by various authors at from 50 per cent to 90 per cent. The opportunity recently presented of observing and successfully operating upon two patients with this most fatal of all abdominal emergencies prompts the recording of their case histories.

CASE I. A 60 year old American housewife was admitted to the Henry Ford Hospital as an emergency July 8, 1935, because of severe abdominal pain. She had been seized with sudden severe generalized abdominal pain thirteen hours previous to admission following the taking of food. The pain was intermittent in character and had gradually increased in severity. Vomiting had been present since the onset and had recurred at intervals of twenty to thirty minutes. The vomitus at first had consisted of gastric contents, but at the time of admission was mostly mucus. The pain was so severe that morphine had been administered by her family physician. There had been no bowel movement since the pain began but she had passed flatus.

The past history had no bearing on the condition. There had been no operations, no suggestion of cardiovascular disease, no previous attacks of abdominal pain.

The abdomen was tense throughout and there was pronounced muscular spasm all over, most marked in the left lower quadrant. There was no distention. The temperature was 97.8°F., the pulse 88, respirations 20. W.B.C. 16,500, with polymorphonuclears 89 per cent. Blood pressure was 118/80.

A tentative diagnosis of an acute surgical condition of the abdomen was made, with twisted ovarian cyst, ruptured peptic ulcer and acute pancreatitis being considered as possibilities. General supportive measures, including the intravenous administration of 10 per cent glucose, were instituted. The persistence of severe pain and continuation of the vomiting suggested intestinal obstruction so strongly that operation was advised.

Laparotomy was performed within twenty-four hours of the onset of the pain. Under spinal anesthetic a midline infra-umbilical incision was made, reflecting the right rectus mucle outward. When the peritoneal cavity was opened, several loops of black gangrenous small bowel presented but there was no undue amount of peritoneal fluid. The gangrenous loops of intestine were not dilated. The involved area of bowel was sharply demarcated at a point 4 inches from the ileocecal valve, and as the intestine was followed proximally, the amount of gangrene decreased until it finally shaded off into hyperemic and finally normal appearing intestine. The mesentery corresponding to the area of gangrenous intestine was

thickened and discolored. A segment of damaged ileum 4½ feet in length was resected. The small stump of terminal ileum was ligated and invaginated into the wall of the cecum with a series of purse-string sutures. A mobile cecum made it easy to do an end-to-side anastomosis of the ileum to the cecum.

Recovery from the resection was extremely smooth but hospitalization was lengthened to a month because of the development of a thrombophlebitis of the right leg.

In spite of the removal of 4½ feet of terminal ileum the patient experienced no frequency of bowel movement. Her general health three years after operation was excellent.

Pathologic Report. The diagnosis was mesenteric thrombosis with gangrene of the small bowel. The proximal end of the artery in the involved area of mesentery was lined with a yellowish membrane but was not completely occluded. A section of the arteries of the bowel wall showed that they were patent but thrombotic masses filled the lumen of the venous channels.

Case ii. A 25-year old American housewife, six months pregnant, was admitted as an emergency to the Henry Ford Hospital October 7, 1935 because of severe abdominal pain. The pain had come on suddenly a few hours previous to admission. It was continuous, radiating into the back and right flank, and of such severity that morphine was required for its control. There was no vomiting. At first the pain was thought to be due to the onset of premature labor, but there were no accompanying uterine contractions.

There was no distention. The abdomen was diffusely tender to palpation and there was slight general muscle spasm, but the pain and tenderness were more pronounced in the right flank. A right rectus appendectomy scar was present. The temperature was 98.8, the pulse rate 84 and respirations 20. The white count was 18,400 with polymorphonuclears 90 per cent. The ureters were catheterized and pyelograms were made because of pain in the right flank, but the results were negative. The following day the pain persisted, but vomiting did not appear until nearly forty-eight hours after the onset of the pain. A diagnosis of intestinal obstruction was made and operation advised.

Laparotomy was performed forty-eight hours after the onset of the attack. Under ethylene

and ether anesthesia, the abdomen was opened through a subumbilical right rectus incision. A large amount of blood stained fluid, estimated at 1,500 c.c. escaped from the peritoneal cavity and an inert mass of gangrenous ileum presented to the right of the uterus, completely filling the wound.

A diagnosis of superior mesenteric thrombosis seemed obvious, but, when an effort was made to bring the diseased loops of bowel up into the wound, it was found that a portion of the involved area was tied down by an adhesion band. This suggested that the band was the cause of the obstruction. However, the extension of the diseased process beyond the area compressed by the band and its limitation to the region supplied by the superior mesenteric artery forced a return to the original diagnosis of mesenteric thrombosis.

The involved area of ileum measured 9 feet in length and its mesentery was also black and gangrenous. The last 4 inches of the ileum were not damaged and there was a sharp line of demarcation between the gangrenous and healthy intestine. The gangrenous ileum and its mesentery were resected. The stump of the ileum was ligated and invaginated into the wall of the cecum by means of a series of purse-string sutures. The cecum was mobilized by incising the peritoneum along the floor of the right paracolic gutter. An end-to-side anastomosis of the ileum to the cecum was then readily done.

Recovery from the abdominal operation was satisfactory but hospitalization was prolonged to five weeks because of the delivery of a stillborn baby on the second postoperative day and the subsequent development of bronchopneumonia.

The patient was inconvenienced by frequent bowel movements for some time but eventually this cleared up and three years after operation, in spite of the loss of nearly one-half of her small intestine, her health was good.

Pathologic Report. The diagnosis of mesenteric thrombosis with gangrene of the small bowel was confirmed. The lumen of the gut was filled with bloody liquid material, the wall hemorrhagic and easily torn. The blood vessels of the mesentery showed thrombi.

COMMENT

The two case histories cited are examples of the truth of the dictum of Boyce and

McFetridge that the most hopeful thing about mesenteric thrombosis is that it can be cured by operation. The outstanding clinical feature of both case histories was the severity of the abdominal pain which required morphia for its alleviation. Vomiting was present in both cases. In Case II it persisted from the onset while in Case I it was a late manifestation of the condition. In both patients, the vomiting was overshadowed by the agonizing pain. This is exactly the reverse of the usual picture of obstructions of the small bowel from other causes, where vomiting is the essential point and pain occupies a secondary position in the symptomatology. The approximately normal temperature and pulse rate negative the presence of infection and the early high leucocytic response indicates tissue death.

Case I is clearly an example of the venous type of mesenteric occlusion, though the terminal mesenteric arteries, while still patent, were secondarily involved.

Case II represents the arterial type because of the rapid onset of gangrene. Section of the diseased mesentery showed that both arteries and veins were involved. The diagnosis of primary mesenteric thrombosis in Case II may be challenged because of the presence of the adhesion band. Points in favor of its inclusion are: The clinical course with late signs of obstruction, non-dilatation of the involved loops of intestine, thrombosis of both mesenteric artery and vein with resulting complete gangrene of the bowel wall and extension of the gangrenous process to the mesentery itself, and finally the pathologic report of mesenteric thrombosis. The finding of a definite line of demarcation between diseased and healthy bowel exactly at the point of anastomosis between the ileocolic and terminal intestinal branches of the superior mesenteric artery appears to clinch the diagnosis of primary mesenteric thrombosis. The presence of the band was probably incidental and of secondary importance.

An interesting and identical verification of a well known anatomic fact was made in both cases. In both instances the terminal 4 inches of the ileum was not involved since this portion of the ileum derives its blood supply from the ileocolic artery and not from the terminal intestinal branches of the superior mesenteric artery. Furthermore, the finding of a mobile cecum in Case I and its utilization for anastomotic purposes suggested the procedure adopted in Case II, viz., mobilizing the cecum and ascending colon and anastomosing the ileum to the cecum rather than subjecting the patient to the further hazard of resection of the ileocecal segment. Resection and anastomosis constituted the procedure of choice as well as of expediency in both cases, because exteriorization would have necessitated mobilizing the entire ileocecal segment. The stump of the terminal ileum was too short to bring up into the abdominal wound.

SUMMARY

Two cases of mesenteric thrombosis are reported. The most outstanding clinical feature was the severity of the abdominal pain. Vomiting was of secondary importance in the symptomatology, thereby reversing the usual picture of small intestinal obstruction. Recovery followed resection of 9 feet of ileum in one patient and 4½ feet in the other.

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A CASE OF PRIMARY LYMPHOSARCOMA OCCUPYING TWO-THIRDS OF THE LUMEN OF THE STOMACH

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IT is generally conceded that the incidence of primary gastric sarcoma is about 1 per cent of all gastric neoplasms.[1,9,10]

Pack and McNeer[1] in 1935 reported nine cases of sarcoma of the stomach, three of which were primary gastric lymphosarcomata. They stated that out of 40,000 cases of benign and malignant neoplasms of all organs treated at the Memorial Hospital, there were 400 cases of malignant neoplasms of the stomach. In that series there were only five cases of sarcoma, two of which were primary gastric lymphosarcomata. They reported a third such case from the New Haven Hospital.

Cheever[2] reviewed 628 cases of gastric tumors treated at the Peter Bent Brigham Hospital, among which there were nine cases of lymphosarcomata.

Cases of general lymphosarcoma which have come to autopsy have not infrequently been found to have secondary metastatic involvement of the stomach. Two such cases were included among those reported by Pack and McNeer.

The first case of sarcoma of the stomach reported in the literature was by Sibley in 1816, quoted by Walton.[3]

Primary gastric lymphosarcoma may occur at any age. Finlayson's[4] patient was 3½ years old. Jones and Carmody's[5] patient was 9 years of age. Di Giacoma's[6] was 65 years old, Gosset's patient cited by Balfour and McCann[7] was aged 85 years.

Sarcoma may grow from any aspect of the stomach. They have been described as intragastric, intramural, and even extragastric in location.

Types of gastric sarcoma reported in the literature are angiosarcoma, spindle-cell sarcoma, neurosarcoma and fibrosarcoma. The latter two are of higher malignancy and occasionally involve the pylorus. Another type called round or alveolar sarcoma is usually infiltrative in character and a less common variety.

Clinically, it is most often impossible to differentiate between carcinoma and sarcoma of the stomach. Symptoms of epigastric pressure, pain, vomiting, hematemesis, tarry stools, anemia and loss of weight are common to both.

Patients between 20 and 35 years of age or less, in whom a large gastric neoplasm is found, most likely are suffering from sarcoma rather than carcinoma of the stomach. In addition to the usual Roentgen examination a gastroscopic examination should be made, preferably with the gastroscope containing the retrograde optical attachment described by Korbsch.[8] This may aid in making a definite diagnosis. Very occasionally a piece of the gastric neoplasm may become dislodged during regurgitation or while doing a gastric analysis. Pathologic examination of the piece of tissue would clinch the diagnosis.

It is generally recognized that when gastric carcinoma can be clinically palpated, it is most often already too late for surgical intervention because of metastases and even direct invasion of the surrounding structures. This is not necessarily so in sarcoma of the stomach. Metastases, when they do occur, appear much later in the disease, and may be present in the perigastric lymph nodes, liver, mesentery, lungs, peritoneum and also in the superficial lymph nodes. When the latter appear, a biopsy may easily confirm the diagnosis.

Lymphosarcoma is radiosensitive. The malignant lymphocytes are sensitive to radium as well as to the Roentgen ray. Desjardins stated that this therapy alone

reported surgically cured nineteen years after partial gastrectomy. This is the longest postoperative cure of this lesion which has been reported.
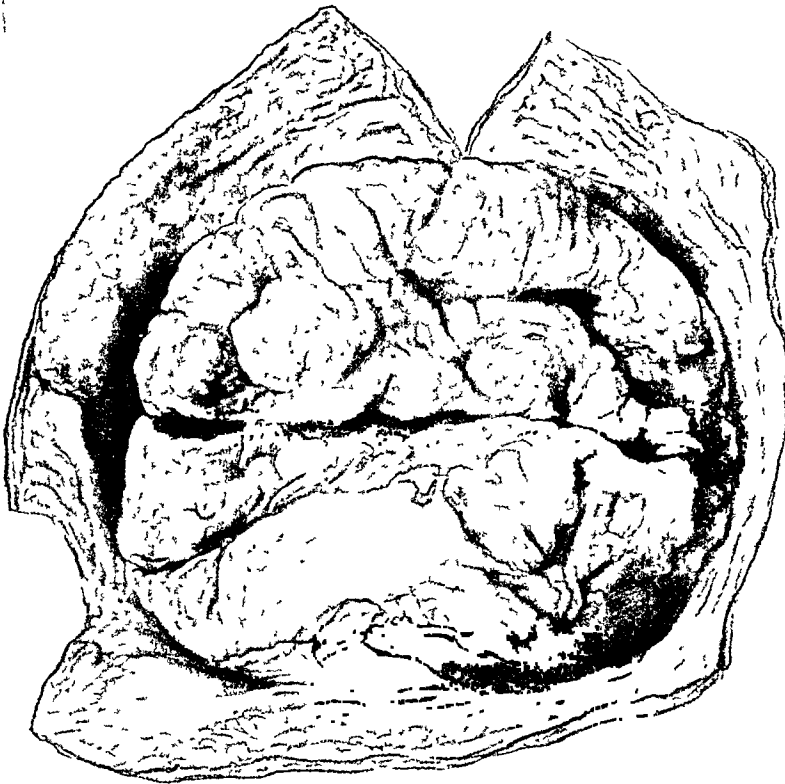
FIG. 1. Excised distal two-thirds of stomach showing tumor size of a baby's head which grew from the posterior wall and occupied its lumen.

might cure early cases of gastric lymphosarcoma. It would, however, appear to be a dangerous procedure to treat the larger tumors by radiation alone because of possible necrosis, hemorrhage or perforation. Drane[11] advises the administration of medium wave length Roentgen therapy.

Most cases reviewed in the literature have not survived operation for more than one year. There have, however, been notable exceptions which have survived and have remained well for many years. Balfour and McCann's[7] patients after gastrectomy lived on the average of eleven months and their longest survival was nine years. Jones and Carmody's[5] case of gastric lymphosarcoma in a boy 9 years old, was

CASE REPORT

M. T., 38 years of age, a Greek woman, was admitted to the Misericordia Hospital on February 8, 1930. She was married and had three children. A cholecystectomy and appendectomy had been performed five years previously at another institution. For four months prior to admission to the hospital she had complained of epigastric cramp-like pains which radiated to the interscapular region. These occurred about twenty minutes after meals and lasted for many hours. Nausea, vomiting and eructations were present. Fourteen days before admission she began to have hematemesis soon after taking food. This persisted for seven days. She had two similar attacks of copious bloody vomiting during the ensuing week. The stools were tarry black in color, but there was no diarrhea. The patient had lost

29 pounds within four months and was becoming progressively weaker.

Physical examination revealed the patient to be acutely ill. There was marked pallor and evidence of considerable loss of weight. The abdomen was protuberant. A large tumor the size of a grape-fruit was easily palpable in the left upper quadrant. It was tender and did not move with respirations, but seemed to be attached to the deeper structures. The mass was firm, irregular and extended behind the region of the spleen. The liver was not enlarged and there was no ascites present. The heart and lungs were clinically negative. No enlarged lymph nodes were palpable. The rectum and pelvic organs revealed no abnormality.

Gastric analysis could not be attempted in the presence of gross hematemesis and danger of perforation of the stomach.

Roentgen ray examination revealed an unusual defect in the pars media and pars pylorica of the stomach. There was no evidence of obstruction. The roentgenologist could not be certain whether the tumor was intragastric or extragastric. Clinically, the lesion was thought to be either carcinoma of the stomach or a pancreatic tumor.

Blood count showed Erythrocytes 3,670,000; white cells 13,800; polymorphonuclear leucocytes 80 per cent; lymphocytes 18 per cent; mononuclear cells 2 per cent. The blood pressure was 116/80. Blood chemistry revealed: urea nitrogen 15; creatinine 1; uric acid 3.4; sugar 106 mg. per cent. Cystoscopic examination and pyelography were negative.

Exploratory operation was performed on February 13, 1930. A median epigastric incision was made. Upon opening the peritoneum no free fluid was encountered. The stomach wall was dilated and hypertrophied. An oval tumor the size of a baby's head was found to be growing from the posterior wall of the stomach and occupying about two-thirds of its lumen. A few metastases were present, only in the regional lymph nodes at the lesser curvature.

A subtotal gastrectomy was performed. The upper third of the remaining part of the stomach was closed and a Polya operation with retrocolic anastomosis with the first portion of the jejunum was done. Moderate postoperative shock followed, which was relieved with glucose-saline infusions and medication. A blood transfusion of 600 c.c. of whole blood was given.

The patient rallied after a stormy postoperative course. Soon after operation she was able to take small quantities of easily digestible
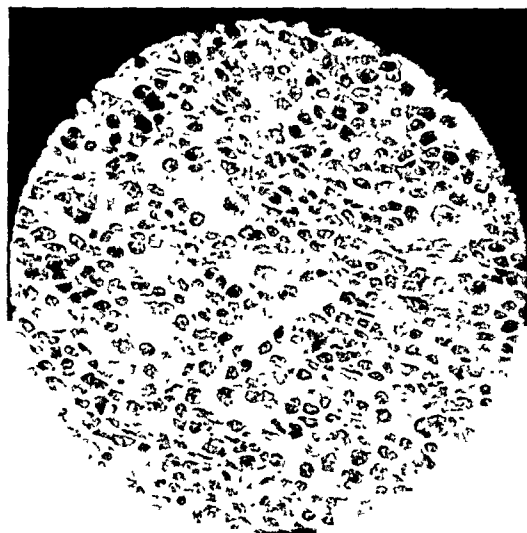
Fig. 2. Photomicrograph of the tumor showing round cells varying in size containing large pale nuclei with numerous mitotic figures. The supporting tissue is scant, delicate and contains few fibrils.

foods, and she became progressively stronger. She was discharged from the hospital on April 20, 1930.

Three weeks after she left the hospital, I was called hurriedly to see her during the night. She complained of severe thoracic pains, dyspnea, cough and hemoptysis, and died suddenly from what clinically resembled pulmonary embolism. No permission for a postmortem examination could be obtained.

Pathologic examination of the portion of the stomach containing the tumor removed at operation was made by Dr. R. C. Schleussner. The specimen (Fig. 1) consisted of a section of stomach measuring 17 cm. × 20 cm. The stomach had been opened along the anterior wall and weighed 580 gm. Arising from the posterior wall was a large ovoid tumor 14 × 9 × 5 cm. It had a broad flat base and its surface was superficially ulcerated. On section it was of uniform grayish white color and was quite friable. The edge of the tumor could be seen to be infiltrating the wall of the stomach beneath the adjoining mucous membrane.

Sections through the main body of the tumor showed that it was composed of round cells about 10 mm. in diameter, with moderate variation in the size of the cells. The nuclei

were large and pale; numerous mitotic figures could be seen. The cytoplasm was scant and irregularly extended in places. There was only a small amount of delicate supporting tissue present, little intracellular brown substance and very few fibrils. The tumor penetrated all coats of the stomach wall and displaced the normal tissue. Numerous tumor cells infiltrated the stomach wall beneath the mucosa at the border of the tumor. The tumor tissue contained many thin walled blood vessels. (Fig. 2.)

SUMMARY

A case of primary lymphosarcoma occupying two-thirds of the lumen of the stomach is reported. This occurred in a woman 38 years old.

Primary lymphosarcoma of the stomach occurs in about 1 per cent of all gastric neoplasms.

The youngest patient with this lesion reported in the literature was 3½ years of age, and the oldest patient was 85 years.

The longest survival after gastrectomy reported surgically cured was 19 years (Jones and Carmody).

Patients with large palpable gastric tumors before middle age should be suspected of having sarcoma of the stomach and not carcinoma.

Subtotal or even total gastrectomy followed by deep Roentgen therapy is the procedure generally followed.

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EPIDEMIC PAROTITIS (MUMPS) AND ACUTE APPENDICITIS

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"EPIDEMIC parotitis is an acute contagious disease characterized by swelling and tenderness in one or more of the salivary glands, by comparatively mild constitutional manifestations and by a tendency in adult males to develop orchitis as a complication."[20] It is caused by a filtrable virus.[11,14] It also may attack the pancreas, mature ovaries, the meninges and the brain. Fatal cases of the meningitic type have been reported.[12] It is not necessary in a case of disease caused by the virus that the salivary glands be involved. Meningo-encephalitis is reported by Montgomery (quoted by Stimson[20]). Orchitis without parotitis has also been reported.[3,9,10]

The incubation period is from twelve to twenty-four days at the probable outside limits.[20]

The disease may be preceded by a slight fever and some chilliness with general malaise and dizziness for twelve to twenty-four hours before the salivary swelling is noted. Pain in and behind the ear and sore throat are also frequent complaints at this time.[20] These symptoms are variable and may be very severe or entirely absent. The duration is from seven days to about three weeks with rare cases extending even longer. Usually the swelling is down in about ten days. Abscess formation does not occur.[20] The duct orifice may be reddened and puffy but normal saliva is grossly present as compared to the pyogenic parotitis of the ascending type with purulent exudate.[7,8] The temperature varies from normal to over 103°F.

Complications in this disease occur usually about four to seven days after the onset. Of these orchitis is the most frequent. This rarely occurs before puberty and is present in about 20 per cent of adults.[20] Sexual activity increases the evidence of orchitis, as demonstrated by French and German authors during the war. They noted lower incidence in soldiers at the front as compared to the incidence at the bases where soldiers had opportunity to gratify their sex desires.[15]

The swelling may be unilateral or bilateral (about 16 per cent).[20] It usually lasts from four to seven days and is accompanied by high fever and occasionally severe systemic reaction. Wolbarst states that the swelling is caused by acute inflammatory reaction in the testicle with areas of groups of seminiferous (convoluted) tubules completely destroyed and distended with exudate. The exudate consists chiefly of polymorphonuclear leucocytes and the phagocytic endothelial leukocytes. The intertubular connective tissue is edematous. The description is taken from report of microscopic sections received during the acute stage by operation.[19]

Atrophy results in about 60 per cent.[19] Sex function is usually regained and complete sterility as an aftermath is exceedingly rare.[20] The epididymis may be involved also. J. D. Rolleston[15] mentions a case of suppuration of the testicle. Here, however, scarlet fever was present at the same time.

Pancreatitis occurs occasionally. It is found more often in adult women and rarely is seen before puberty. It begins about a week after the onset and is characterized by prostration, severe nausea and vomiting and epigastric pain. It usually subsides in four or five days with complete recovery. Sugar is not found in the urine at the time.[20] Possibly with the

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blood amylase tests this might be more accurately determined. Rolleston[15] states that diabetes has been reported to follow mumps.

Inflammations of the thyroid, mammary, lacrimal glands, ovaries and prostate also have been reported. The kidneys may show a mild form of diffuse nephritis. Meningo-encephalitis is not very rare. It may vary from a mild form to the severe type which may show signs of encephalitis with delirium and coma and may even be fatal.[12] The spinal fluid shows increased pressure, presence of globulin and a lymphocytosis with from 50 to 1,000 cells. Usually complete recovery occurs after a few days. Deafness may occur due to nerve involvement. This is often permanent.[20]

Simonin in 1903[16] first reported the occurrence of acute appendicitis and epidemic parotitis. Since then cases have been reported by Jaloquier (1909), Gauchier and Swynghidow (1925), Benassi (1927), Sandler and Finne (1932), and Seelye (1935). Undoubtedly many other cases have been reported which were not noted by the writer as well as many, probably the greater number, which have not been reported at all. Dr. George F. Dick in the Yearbook of Medicine for 1935 notes the increase of appendicitis in epidemics of mumps.

Rosenau and Dunlap reported an epidemic of appendicitis and parotitis at the Culver Military Academy in 1916.[18] There were thirty four cases of parotitis and eight cases of appendicitis. However, the two conditions did not occur in the same individuals. They isolated a streptococcus which gave rise to parotitis and appendicitis in varying proportions in rabbits. In view of the recent work by Johnson and Goodpasture proving that a virus is the cause of mumps, the inference drawn that both conditions were caused by the same organism, a streptococcus, seems questionable.

The few articles obtained on the subject of mumps and appendicitis mentioned Benassi[2] as feeling that the cause of the mumps was also the cause of the appendicitis. His opinion is based on one fatal case occuring in a boy of 18 who developed a bilateral orchitis on the third day after the parotid swelling. Abdominal pain began on the fifth day and continued until his admission to the hospital about two weeks later. At this time he showed signs of advanced peritonitis and died without being operated on. A ruptured appendix with diffuse peritonitis was found at the autopsy. Benassi states that "the appendicitis developed at the height of the parotitis and with some doubt and reserve there is enough evidence to support the following conclusion, that the virus or organism which lodges in Stenson's duct travels to the appendiceal region by way of the digestive tract or by way of the hematogenous or lymphatic system."

Appendicitis in the cases cited began within a week after the onset of the swelling. Pancreatitis, oöphoritis, etc., also begin about the same time. All these would tend to support the idea that there is some connection between epidemic parotitis and acute appendicitis. The few cases reported compared to the number of cases of mumps in a community, however, contradict this hypothesis and would tend to suggest coincidence rather than cause. This is borne out by the medical profession of this vicinity, most of whom have never seen both conditions in the same patient.

Abdominal pain is seen in other of the acute contagious diseases such as scarlet fever,[20] measles, and in anterior poliomyelitis. Appendicitis occurs occasionally during the course of any of the contagious diseases. A direct connection is not generally accepted between the systemic condition and the appendicitis with the exception of tonsillitis and grippe.

CASE REPORT

In March, 1935 the writer was called to see a boy of 14 with acute swelling of the left parotid gland of three days' duration due to mumps. He had been taken with acute pain in the right

lower quadrant twelve hours previously. His temperature was 100 rectally, pulse 88, respirations 20. The abdominal pain came on suddenly without vomiting and with gradual localization over McBurney's area. The pain was severe and continuous.

The past history was negative and physical examination also proved negative aside from the swollen parotid gland and marked muscle spasm and rebound pain over McBurney's area.

A diagnosis of acute appendicitis was made and operation performed under local anesthesia with a few moments of gas and oxygen on opening the peritoneum. There was a moderate amount of cloudy fluid in the abdomen. The appendix was markedly enlarged, covered with fibrin and acutely inflamed. Culture from the abdominal cavity showed Gram-negative bacilli resembling diphtheriods. The microscopic diagnosis was acute catarrhal appendicitis and peri-appendicitis. Convalescence was uneventful.

The second case was seen January 6, 1938, at midnight. The patient, a boy aged 19, had been complaining of pain in the right lower quadrant off and on for about four weeks. It was crampy in character and was not accompanied by nausea and vomiting. During the four weeks period he had also noticed tenderness in the right testicle and stated that his underwear felt tight. He denied any swelling or discharge. There was no history of exposure, and he had no chills or night sweats. During the previous day the pains had become more severe although they were similar to his previous attacks. No frequency or dysuria was reported.

Examination showed muscle spasm and some rebound pain over McBurney's area. The abdomen was otherwise soft and normal. The right testicle was not swollen, but pulling the testicle down elicited pain on the right side. This, however, was not the same pain as that for which attention had been sought. The urethra was normal in appearance and there was no discharge. White count and urinalysis were normal. Despite the negative urinary complaints a flat plate was done the following morning which was negative for stones.

The pain still persisted but the temperature and pulse were still normal. At operation the appendix grossly showed moderate congestion. Postoperatively the temperature rose to 104 although the patient did not look at all ill. On the second day, the temperature was 102. At this time swelling of the right testicle manifested itself. The testicle was elevated and ice caps applied. On the third day the right parotid gland showed a swelling about the size of a large walnut just in front of the ear. Stenson's duct was slightly injected and on pressure clear saliva was noted. The swelling of the gland subsided in two days with the application of ice caps. The temperature fluctuated between 100 and 103 until the sixth day when it dropped to normal. The swelling of the testicle subsided in about a week.

One month after the operation there was definite atrophy of the right testicle. A detailed microscopic examination of the appendix showed evidence of an acute exacerbation of a chronic appendix. The mucosa was destroyed in many areas and there was pus in the lumen. It is difficult to believe that the patient's pain over a period of a month was due to appendicitis. At the same time, however, the period is also rather long for beginning parotitis. This case might be considered as in favor of Benassi's hypothesis.

In a discussion of these cases the question arose whether the second case was actually one of epidemic parotitis. From my past experience[7,8] I cannot differentiate the appearance of the gland and duct orifice from that seen in the hematogenous type of postoperative parotitis. However, I have not noted an orchitis coincidental with the parotitis at any time nor have I been able to find such cases in the literature. Furthermore, atrophy of the testicle due to or coincidental with postoperative parotitis has not been reported. Aside from cases of severe systemic infections, such as typhoid, I find no causes of atrophy of the testes mentioned by Young[21] except mumps, excluding, trauma or pyogenic infection where abscess occurs. Furthermore, in Binghamton we had at the time a widespread epidemic of parotitis with a large number of cases of orchitis in adults. This boy was exposed to mumps and stated that prior to this attack he had never had the disease.

It is my impression that this patient had a low grade infection of the testicle

due to the virus of mumps without salivary gland involvement. It is possible that the anesthetic (ether) and the trauma incidental to operation caused a flare-up in both the parotid gland and the testicle with evident gross pathology. The comparatively mild course of the disease and resulting atrophy of the testicle would tend to substantiate the mumps etiology.

SUMMARY

Two cases of acute appendicitis and epidemic parotitis have been reported. A general survey of the literature and a careful analysis of these cases do not tend to substantiate the theory of Benassi that epidemic parotitis and acute appendicitis are caused by the same organism. It is felt that the condition when found is coincidental.

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RETROPERITONEAL POSITION OF THE ASCENDING COLON

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A CAREFUL search of the literature fails to reveal a case with findings similar to those recorded in this paper, namely the retroperitoneal position of the ascending colon in an adult. Developmental defects and malpositions are frequently met, especially in infants; but many of the bizarre and altogether strange cases pass by unnoticed and consequently remain unreported, because these patients do not have the benefit of radiography or do not come to operation or autopsy.

Among the congenital findings, previously mentioned by other writers, particularly referable to the colon are:

1. Complete or partial absence of the colon.

2. Transposition of the viscera, which may be partial or complete.

3. Stenosis or atresia.

4. Microcolon.

5. Megacolon.

6. Double-barreled colon.

7. Intestinal fistula.

8. Termination of the gut in an umbilical hernia.

9. Diverticula.

10. Associated abnormalities of the mesentery and peritoneum.

It is the consensus of opinion that developmental errors involving the gut are frequently found in babies and children, who often present other congenital anomalies, such as cleft palate, atresia of the urethra, anus or vagina; spinal cord defects causing fecal and urinary incontinence, etc. Malformations and irregular positions of the gut are said to occur frequently in males.

On the evening of April 8, 1938, Miss H. H., 34 years old, was admitted to the hospital. Three days previously she had been taken ill quite suddenly with cramp-like, generalized abdominal pain which occasionally let up, but returned with greater severity. The pain gradually seemed to localize in the lower left quadrant, and upon the day of admission its severity was intense. There was no history of vomiting, though the patient complained of considerable nausea and had eaten little for three days. The day before and on the day of admission she had had no bowel movement. An enema produced no results. The patient had noticed a change in the size of the abdomen shortly after onset of the first abdominal pain. This increased to marked distention upon admission.

About two years before she had been in the hospital for observation for a short period of time, because of some vague intestinal complaint. Otherwise her general health had been fairly good. Her menstrual periods were regular; the last one had occurred two weeks previous. An accident had caused loss of sight in the right eye in childhood. The family history was positive for diabetes mellitus and cardiac disease.

Upon admission temperature was 101 degrees, pulse 140 and respiration 26. The abdomen was markedly distended, approximately to the size of an eight months' pregnancy. Tenderness could be elicited upon palpation of any portion of the abdomen, the point of greatest pain being to the left of the umbilicus. Peristalsis could not be heard. The laboratory findings were quite normal.

The clinical picture apparently pointed to an intestinal obstruction, in spite of the absence of vomiting and the normal blood count. The roentgenologist verified this diagnosis, and operation was deemed necessary. The patient was operated upon that night under spinal anesthesia.

A midline incision from the umbilicus to the symphysis was made. Opening of the peritoneum produced an overflow of free serous fluid. The cecum lay in the midline and was enormously distended, to approximately six times its normal size. Once out of the abdomen it could not be replaced through the incision

due to its enormous distention. Its coat, as well as that of the greatly distended small intestine, was a dark red. In searching for the point of obstruction it was discovered that the cecum terminated suddenly to the right of the midline and that the transverse colon appeared to rise as mysteriously from the region of the hepatic flexure with no evidence of a connecting ascending colon. The descending colon and sigmoid were normal in size and position. The stomach and gall-bladder presented no pathology. The uterus, ovaries and tubes were definitely normal. These observations were made in a few moments. The actual cause or point of obstruction could not be located, but it seemed advisable to close the abdomen as quickly as possible at this time and relieve the distention by an emergency cecostomy, because of the exceedingly poor condition of the patient who went into shock on the table. Moreover, it did not seem to be good surgery to handle further the greatly distended gut for fear of traumatizing it. Further investigation could be carried on at a second operation should the patient recover.

A cecostomy was performed by bringing the appendix through a stab wound and amputating it. The abdomen was closed in layers. The patient rallied after a blood transfusion and continuous intravenous glucose. After the second day postoperatively she showed marked improvement. The colostomy functioned well. In fact the patient appeared to grow progressively better until the eleventh day, when temperature, pulse and respiration slowly mounted. Her general condition suddenly changed on the twelfth day, when she died at 10:25 P.M.

The pathologist found at autopsy that the cecum was in the midline, with a well walled-off cecostomy opening in the stab wound on the right side. The elongated, but now not distended cecum dipped down through the mesentery to the right of the midline and appeared to end there. The transverse colon appeared to spring from the region of the hepatic flexure, but showed no attachment at this point.

Careful dissection and freeing of the mesentery and areolar tissue coverings showed the ascending colon to be entirely retroperitoneal, lying at its beginning proximal to the vertebral column, assuming then an almost horizontal direction parallel to the transverse colon.

In its distal third it took a tangential direction to the hepatic flexure. A fibrous band attached to the spinal column bound the gut at its proximal end, forming a complete obstruction. The gut immediately beyond this point had the shape of a half twist.

The apparent break in the continuity of the ascending and transverse colon previously noted at time of operation, was due to the fact that the ascending colon grew out of the peritoneal cavity. Both transverse and ascending colon were normal in position.

This defect may be explained most satisfactorily upon the basis of a defect in the prenatal course of torsion and mesenteric fusions. About the middle of the fourth month, all that portion of the mesentery which is attached to the segments of intestine destined later to become jejunum, ileum, ascending and transverse colon, is attached to the median line of the dorsal wall of the abdomen throughout its entire length. However, the original picture becomes greatly changed, because of fusions which occur in certain portions. The first of these is brought about by the twisting of the primary loop of the intestine. As a result the ascending and descending colons are brought close to the lateral walls of the abdominal cavity. The parietal peritoneum lining the right abdominal wall becomes fused to the right layer of the mesentery attached to the ascending colon. A similar fusion takes place in the left. Thus the ascending and descending colons lose their mesenteries and have a permanently fixed position.

The transverse colon passes ventral to the duodenum. Its mesocolon never comes in contact with the parietal peritoneum, and its line of attachment is transverse to the body wall. This line of attachment forces the duodenum against the dorsal abdominal wall, because of its passage in front of the gut, thus making this part of the gut retroperitoneal. What was the dorsal mesentery disappears and changes into subserous areolar tissue.

The cecum and appendix are primarily a lateral outpouching of the intestines, are

completely encased by peritoneum and do not possess any mesentery.

With the foregoing embryologic facts in mind, two possible theories may be advanced to explain the retroperitoneal ascending colon: (1) Since the transverse mesocolon, through its transverse line of attachment passing across the duodenum, presses this organ against the dorsal abdominal wall causing it to lose its mesentery and become retroperitoneal, can it not be assumed that this same mesocolon could have pressed, in an error of torsion and fusion against the ascending colon (which in this instance lay part way parallel to the duodenum), converting its mesentery into subserous areolar tissue and causing it to be retroperitoneal. (2) It is possible that the ascending portion of the primary gut in its torsion did not stop at a 90 degree angle of the normal colon, but continued torsion until it lay in a nearly transverse direction behind the root of the mesocolon.

At best these are theories, but they give some explanation of the anomaly described.

SUMMARY

1. The purpose of this paper is to report an unusual anomaly of the ascending colon in an adult female who showed no other congenital defects. Anomalies of the gastrointestinal tract are usually found in male children who show other defects.

2. The ascending colon was not in the peritoneal cavity. For this reason the point of obstruction which was in the ascending colon was not found at operation, but later at autopsy.

3. Its retroperitoneal position can probably be explained upon the basis of a defect in the torsion of the primary gut, or defect in the fusion of its mesentery.

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FASCIA LATA TRANSPLANT FOR PTOSIS*

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IT is not the purpose of this paper to encompass an involved discussion on the various operative procedures which
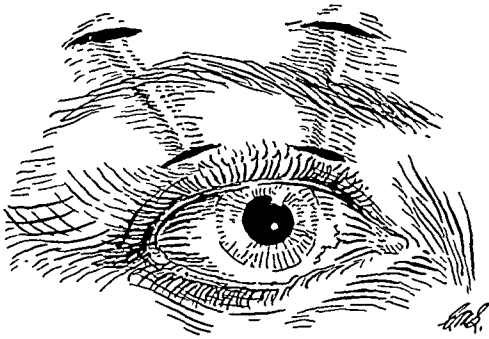
FIG. 1. Method utilized by Lexer employing two strips of fascia.

have been and are being used for the correction of ptosis, but merely to set forth a method I have found satisfactory. The methods any particular surgeon utilizes in carrying out operative procedures depend primarily on his training and the type of surgery he does. Thus, an ophthalmologist will prefer one of the various eye muscle procedures, while the general reconstructive surgeon will probably select a method akin to the one here described. In either case, results form the criteria by which the operation can be judged.

Most of the ptosis cases requiring surgical interference are congenital and usually bilateral. The muscular apparatus of the eye is generally otherwise normal, and this fact forms the basis for the utilization of the superior rectus in various operations described by ophthalmologists. Syphilis and previous encephalitis must also be considered as etiologic factors.

In 1879, Dransart devised the plan of uniting the tarsus to the occipitofrontalis by three double silk sutures which, when withdrawn, left trails of subcutaneous scar,

thus fixing the tarsus to the frontalis.[1] It was desirable in this procedure to allow a certain amount of irritation and perhaps suppuration to promote additional scarring. The obvious weakness of this procedure is that scar is not a very dependable connecting tissue.

Pagenstecher and Hess also utilized threads to connect the tarsus to the occipitofrontalis. Panas modified this procedure by utilizing a pedicled skin flap to connect these two structures.

Lexer, in 1923, employed two strips of fascia (Fig. 1) to make the connection between the tarsus and frontalis. This was a distinct advance in that living tissue was utilized. The fascia lata to a great extent remains unchanged, except for some contraction which emphasizes the lifting result desired.[2]

Derby proposed the use of fascia lata in the form of a sling in 1928 and reported good results.[3]

Blair, Brown, and Hamm have utilized a fascia lata sling from the occipitofrontalis to the upper edge of the tarsus. They recommend the use of two small incisions in the lid through which the fascia is threaded.[4] (Fig. 2.)

The operation, which I have used in the three cases which are presented in this discussion, combines the advantage of the fascia lata sling with resection of a portion of the lid. It is interesting to note at this point that one patient in this series actually suggested some form of suspension from the forehead, inasmuch as he had for years utilized a strip of adhesive running from the forehead to the ptosed eyelid. Nature points the way to some such procedure by overdevelopment of the occipitofrontalis due to a constant attempt to use this muscle in lifting the ptosed lid. This
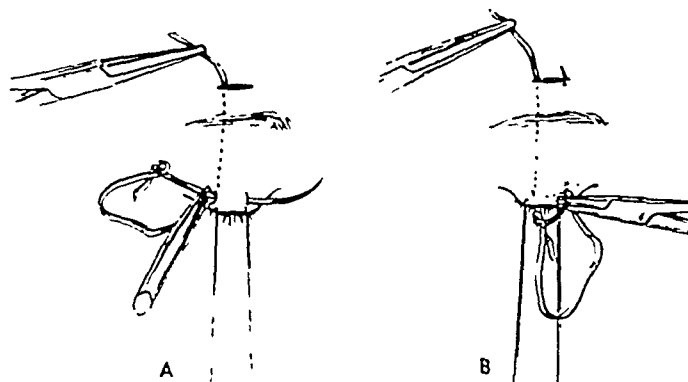
FIG. 2. Blair's method of threading fascia lata. (From Blair, in *Arch. Ophth.*, 7: 831, 1932.)
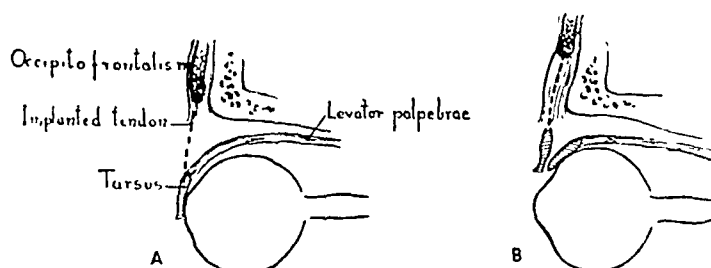
FIG. 3. The normal action of the levator contrasted with that of the transplanted fascia. (From Blair, in *Arch. Ophth.*, 7: 831, 1932.)
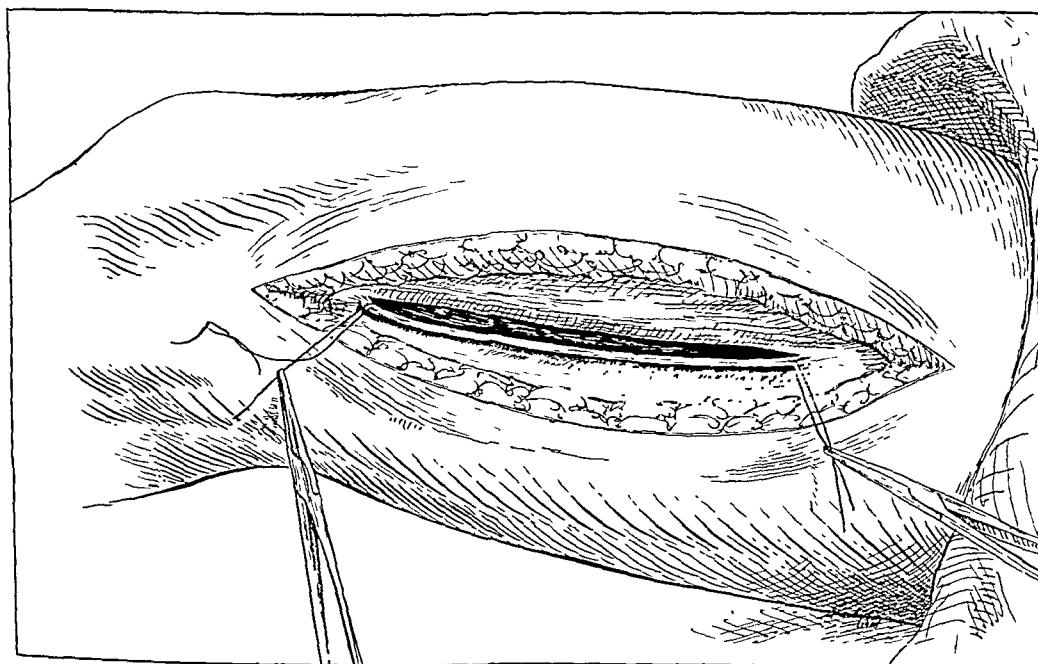
FIG. 4. Selection of strip of fascia lata.

hypertrophy is probably more marked where there is slight residual lifting power in the levator palpebrae superioris.

one functional disadvantage to this procedure. He has shown that the normal functioning levator pulls the tarsus around
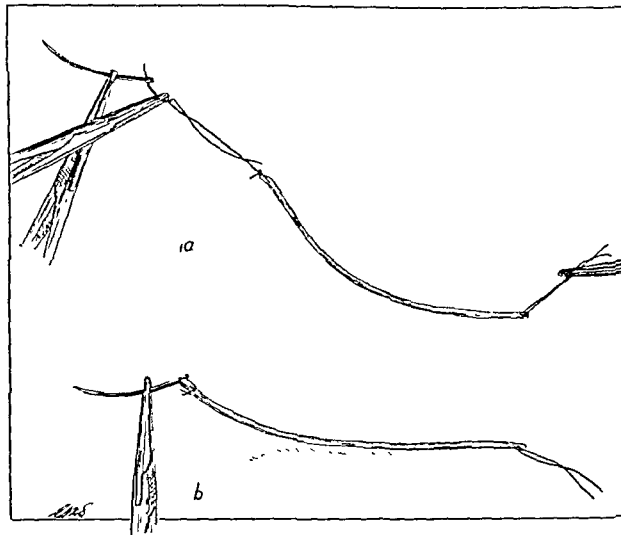
FIG. 5. Method suggested for threading fascia lata.
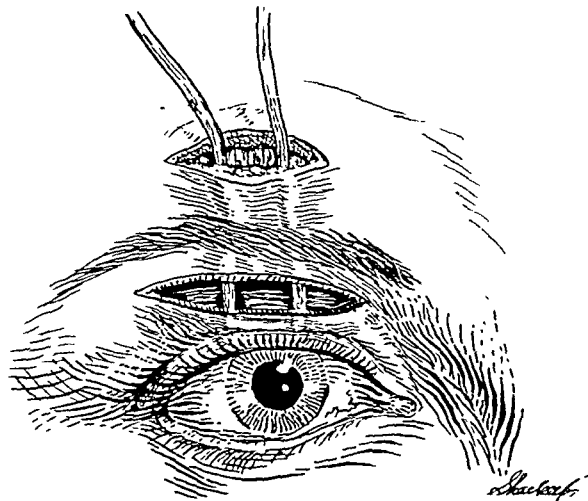
FIG. 6. Utilization of fascia in the form of a mattress suture.

In my experience this combined procedure has been successful both as to function and cosmetic result. Psychologically these people lose their shyness and backwardness. They also lose the constant strained expression when attempting to open their eyes, and most strikingly they lose the necessity for throwing the head backwards in order to be able to see.

Few operations which are reconstructive in scope can claim perfection in imitating nature's own efforts. Blair has pointed out

the circumference of the globe. However, the pull of the occipitofrontalis is directly upwards throughout its entire excursion. (Fig. 3.) This difficulty is more theoretical than real since the ordinary onlooker does not notice any difference.

PREOPERATIVE CARE

Preoperative preparation consists of thorough shaving of the eyebrow on the side to be operated. The thigh on the same side is shaved and washed with ether, soap,

water, and alcohol the night before. It is then wrapped in sterile towels.

The patient is given 1½ gr. of nembutal

The needle is left attached at one end and the strip disconnected.

A half-curve surgical needle, long enough
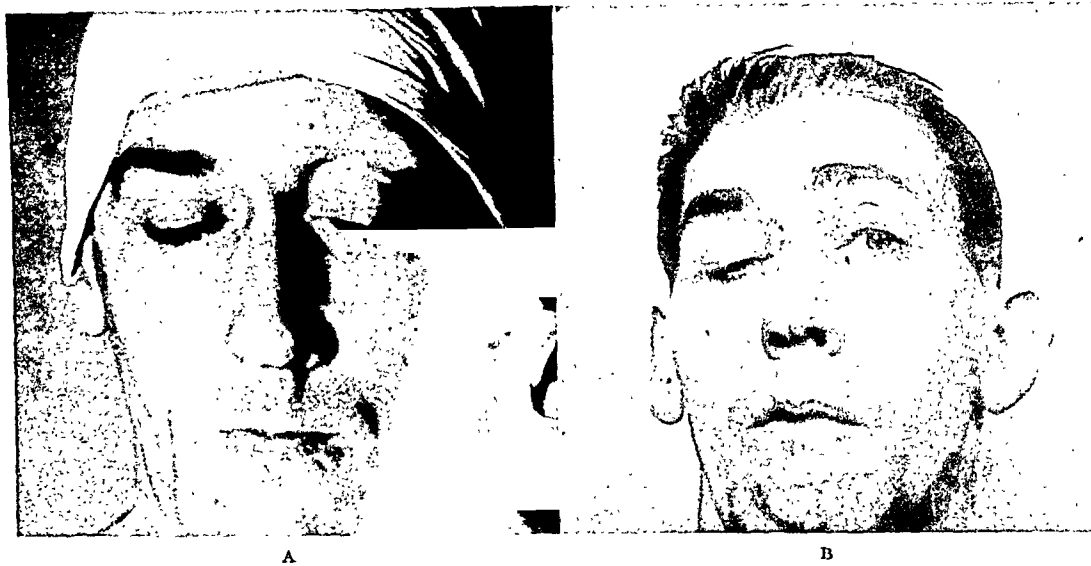
FIG. 7. Case I. A, before operation. B, after operation on left eye.

one hour before and 1½ gr. one-half hour before operation. If the patient is not narcotized at the time of operation morphine sulfate gr. ⅙ is given.

In the operating room, the entire face is thoroughly washed with soap and water for five minutes, and then with 70 per cent alcohol. After thorough drying the operative field is painted and the patient is draped.

The subcutaneous tissues of the upper lid and corresponding supraorbital region are thoroughly infiltrated with 2 per cent novocaine. Attention is then directed to the thigh. After novocaine infiltration an incision is made on the anterolateral surface of the thigh and deepened down to the fascia. The tissue over the fascia is gently cleaned off and a strip of fascia about 15 to 16 cm. long and 3 mm. wide is selected. It is marked out (in the manner suggested by Blair): the longitudinal strands are separated by the back of a bistoury blade and the transverse fibers incised with a No. 15 Bard-Parker blade. The strip is left attached at both ends and a piece of .004 DeKnatel silk is passed through each end with a No. 2 Sheehan needle. (Fig. 4.)

FIG. 8. Case I. Final result.

to reach from above the eyebrow to the tarsus is selected and the fascia threaded on to it by passing the Sheehan needle through

Fig. 9. Case II. A, At rest, before operation. B, at rest, after operation.

Fig. 10. Case II. A, before operation, showing maximum lift. B, after operation, showing maximum lift.

the eye of the larger needle and pulling the fascial strip through. The fascia is then fixed upon itself with the same silk suture.

A   B

Fig. 11. Case III. A, before operation. B, after operation.

(Fig. 5.) The fascial strip is then replaced in the depths of the thigh incision and covered with a pack moistened with saline.

Attention is again directed to the eye. Two fine silk sutures are placed at the lid margin to act as tractors. An oblong portion of lid and orbicularis oculi is excised with a No. 15 Bard-Parker blade. This portion is usually about ¼ inch at its widest portion and tapers toward each end. A transverse incision ½ inch in length is made immediately above the eyebrow and corresponding to the mid-portion of the lid incision. The incision is deepened down to the occipitofrontalis muscle. The subcutaneous tissues are freed down to the eyelid with a dissecting scissors.

The fascia lata is secured and passed as a mattress suture beginning at the medial end of the forehead incision, passing through the muscle and over the supraorbital ridge subcutaneously, to emerge at the medial border of the lid incision. It engages the superior border of the tarsus transversely. A wide enough area must be engaged so that the lid does not become angulated at the point of attachment.

The assistant lifts the lid at this point by pulling on the traction sutures so that the suture is not passed through the conjunctival surface of the lid. The needle is passed upward subcutaneously and then through the occipitofrontalis lateral to the point where the suture first entered the muscle. (Fig. 6.) Here it is tied in one knot and pulled taut until the lid is raised to the point where only about ⅙ of the pupil is covered. At this point the traction sutures are removed. A mosquito forceps is clamped at the knot while interrupted sutures of fine silk are taken to secure it to the occipitofrontalis muscle and also to keep the knot firmly tied. The redundant fascia is excised and both skin incisions are closed with interrupted .0025 silk. A cotton pad is used on both eyes for twenty-four hours.

The fascia lata is closed with a continuous suture of No. 0 chromic and the skin closed with interrupted dermal.

It has been my experience that the conjunctival surface which has not been exposed to the outside for many years may show evidence of irritation twenty-four to forty-eight hours later. If so, simple boric acid eye irrigations and argyrol instillations are of value.

The facial sutures are removed in three days and at this time the patient may start to lift his lid voluntarily. Edema is present for forty-eight to seventy-two hours, but this rapidly subsides. At the end of three weeks additional contraction has usually raised the lid a few more millimeters. The skin incisions are barely visible after complete healing has taken place.

CASE RECORDS

CASE I. A. H., laborer, had always had great difficulty in raising his lids. The droop was more marked on the left side, but he was unable to see from either eye without throwing back his head. There was no personal or family history of syphilis. Blood Wassermann was negative.

Physical examination showed bilateral ptosis, more marked on the left. There was definite weakness of the superior rectus on both sides. On the right side the lid could clear half the pupil with effort. On the left side there was barely any lift.

On September 9, 1936 the left lid was operated on. Following operation there was marked conjunctivitis and photophobia. On February 23, 1937 the procedure was repeated on the right side. All wounds healed by primary intention. (Figs. 7 and 8.)

CASE II. J. O'L., age 44, printer, was committed to Mayview City Hospital eleven years previous to operation for repeated attacks of unconsciousness diagnosed as grand mal epilepsy. The patient had poliomyelitis at 3 years of age and dated his ptosis from that time. There was no familial history of insanity, epilepsy, or ptosis.

Physical examination showed a ptosis of the left lid with atrophy of the levator palpebrae superioris. Other eye muscles were normal. Both pupils reacted well to light and accommodation. Blood Wassermann was negative.

Neurologic examination showed an absent knee jerk on the right and atrophy of the left leg muscles. On December 11, 1936 operation was performed on the left lid. All wounds healed by primary intention. Six months later patient was able to lift lid well and the cosmetic result was very satisfactory. (Figs. 9 and 10.)

CASE III. W. C., age 20, a machinist, stated that ever since birth he had been unable to raise his lids. His field of vision was always just about one-half of what it was when he would raise both lids with his fingers. In order to see straight ahead he was forced to throw his head backward. This inability to see properly made him self-conscious and shy and definitely interfered with the proper pursuit of his occupation. There was no history of any similar complaint in his family.

Physical examination showed bilateral atrophy of the levator palpebrae superioris muscles with an inability to raise either lid. Both pupils were partly covered by the lids. Reaction to light and accommodation was normal.

On August 10, 1936 the right lid was repaired and on September 28, 1936 the procedure was repeated on the other side. The resultant functional and cosmetic results were good on both sides. All wounds healed by primary intention. (Fig. 11.)

CONCLUSIONS

This operation is not perfection, but the functional result is excellent and the cosmetic result good. Certain disadvantages have been pointed out, but they are more theoretical than real.

Patients with ptosis should be given the benefit of some satisfactory procedure since the entire psychological make-up is favorably influenced by a good result.

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ECTOPIA AND INGUINAL HERNIA OF OVARY

REPORT OF A CASE OF CONGENITAL ECTOPIA OF OVARY AND FALLOPIAN TUBE IN LABIUM MAJUS

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THE patient here reported was a white female, age 26, married, primipara of healthy and medium constitution, without familial or personal antecedents of note.

At the age of 7 years, she noticed a small tumefaction in the left inguinal region; its size was that of a small marble and it continued to grow with body development. Menstruation began at the age of 14 years. It was regular and free of pain for two months. External reduction of the tumor was attempted after the second menstruation, and for five months the mass could not be detected. During this period, there was cessation of menstruation. When the tumor reappeared, it occupied a lower position in the inguinal region, reaching to the labium majus. Shortly after the tumor reappeared the menstrual flow returned, and has since been regular, of four weeks' interval and four to five days' duration. Instead of a painless menstruation, as that of the first two periods, the patient had pain in the left inguinal region and labium majus, the pain coming on two or three days prior to the appearance of the menstruation and with an increase in the size of tumor. Such a periodic cycle and increase in size of tumor continued until she came to surgery.

Five years before I saw the patient, a truss was applied and worn for two months. Wearing the truss produced much pain with no change in the size and position of the tumor.

The physical and laboratory findings were essentially normal except for a swelling in the left lower inguinal region and labium majus. The main axis of the tumor was oblique downward, extending well into the upper fold of labium majus. It was the size of a mandarin. The skin region was sound and gliding over the tumefaction. The mass was not painful to palpation, had a clear outline of hard elastic consistency, with an area of hardening at the lower pole, but for the main, the tumor was smooth and firm.

Vaginal examination was normal except for a moderate retroversion of the uterus and a tilt of the uterus to the left pelvis. The right adnexa was palpable and in close proximity to the uterus. The left adnexa, due to the lateral position of the uterus to the left inguinal fold, could not be outlined. Attempt to replace the uterus from the left inguinal region position produced radiating pain along the canal of Nuck to the mass in the labium majus.

The diagnosis made was tumor of the left inguinal region and labium majus, probably an ovarian hernia with incarceration.

Operation was performed on August 9, 1937. A preliminary injection of morphine and scopolamine was given, followed by local anesthesia of novocaine and ephedrine. The skin incision was made from the labium majus upward in an oblique manner over the canal of Nuck, for a sufficient distance to assure good exposure. The incision was carried through the skin and superficial fascia. A large globular mass, enclosed within a sac, was found. It was hard in consistency and fairly regular in outline, except for the lower pole and many old adhesions about the sac and the integument. The hernia sac was covered in part by strands of muscle tissue running along the outer lateral surface.

The sac was incised and its content was found to be ovary, fallopian tube, mesovarium and round ligament. The ovarian mass was adherent and fixed to the base of sac along the external and lower portion. The ovary was enlarged and there was a hypertrophy of all structures except the fimbriated end of the fallopian tube. The tumor mass was rotated 180 degrees on its attached pedicle. The tumor appeared dark, apoplectic in color. Also there was hypertrophy of the structures of the pedicle, including the genito-inguinal ligament. The hypertrophy of the pedicle was so great that is was impossible to pass a probe along the canal of Nuck to the internal peritoneal ring. There appeared a definite fibrous sac encircling the mass, extending from the base of ovary upward along the pedicle. This was enclosed in the common

vagina. The canal of Nuck was opened and explored to and above the external peritoneal ring, with excision of the peduncle and removal

mass served as an attachment for the oviduct which was 6 cm. long and .9 cm. in greatest diameter. This was free of gross changes. On the

FIG. 1. Enveloping membrane at base (tunica vaginalis). Gubernaculum (or round ligament) attached to inner wall of tunica and ovary with oviduct at upper margin with hypertrophy of round ligament.

FIG. 2. Cut section, showing opened corpus luteum at upper pole. Several old hemorrhagic follicles are visible and an occasional enlarged follicle.

of the hernia mass. Care was exercised in the excision of the pedicle to avoid hemorrhage. Its structures were separated from the genito-inguinal or round ligament and ligated. The raw surface of the stem of the pedicle was covered over by infolding the loose accompanying areolar tissue. The stem was permitted to drop into the peritoneal cavity. The round ligament was found to be very short, and to release the uterus from the left inguinal canal, we deemed it necessary to excise the genito-inguinal ligament. Its stump was later anchored to the fibrous portion of the abdominus muscle at a point to release the traction on the uterus. Hernioplasty was done by a modified Bassini type of operation. The sutures were placed in an interrupted manner for the shelving layers. The skin was closed with interrupted dermal sutures. The convalescence was normal. The patient remained in bed two weeks. .

Pathologic Examination. Specimen consisted of an ovary 8.5 by 2.5 by 2 cm. Attached to one side of the ovary was a thick meso-ovarium (5.5 cm.) of triangular shape with its base against the ovary. One side of the triangular

opposite side of the triangular mass there arose from the pole of the ovary a rounded, cord-like structure 2 cm. long and .9 cm. in diameter. This, on section, showed a central group of tubular structure, obviously blood vessels. The basal or ovarian end of the cord was continuous with an enveloping pouch of fibromuscular tissue, which was lined by serous membrane similar to that continuous with the surface of the ovary. At the distal end of the triangular mass above mentioned, the oviduct and round ligament approximated each other. At this end the meso-ovarium, round ligament and oviduct had been cut across in the removal of the mass.

On section, the ovary contained at its tubal end a recently formed corpus luteum 2 by 1 cm. Through the remainder of the ovary there were three old cysts 1 by 6 by 7 mm. in diameter, filled with old brown, bloody coagulum. The ovarian capsule was thick and tough and there were a few enlarged follicles near the capsular surface.

Sections showed a dense fibrous capsule and a fibromuscular stroma richly supplied by blood vessels. The veins were dilated and engorged.

A number of enlarged follicles were present and some contained ova and a clear serous fluid. One large follicular cyst was filled with old blood clot. About the margins there was a loose clear connective tissue, in which were entrapped granules of blood pigment. There were also occasional small corpora albicantia which showed similar deposits of the pigment. None of the sections showed lutein cell formation.

COMMENT

Hernia of the ovary is rare, and 95 per cent of ovarian hernias are inguinal. The persistence of the canal of Nuck bears the greatest significance in the causation of inguinal hernia. Normally the canal of Nuck becomes obliterated by the end of the eighth month of intra-uterine life. In a small percentage of cases the canal of Nuck does not become obliterated but remains in part or completely pervious throughout life.

The gubernaculum of the ovary, which is the anlage of the ovarian and round ligament, becomes attached to the cornua of the uterus. This prevents the descent of the ovary into the inguinal canal in normal development, even though the distal end of the gubernaculum is attached to the labium majus.

All writers, in discussing the cause of the descent of the ovary into the inguinal canal (or labium majus), agree that it is a congenital anomaly. The following explanation more nearly approaches the physiology of ovarian hernias: (1) The genital deformity is an indication of absence or deficiency of a given stimulus, perhaps hormonic, to the development of the genital system, which is inclining toward the feminine; it would be in such a case a deviation of development to the male type, which would bring the abnormal descent of the ovary. (2) The failure of the genito-inguinal ligament to adhere to the deformed or undeveloped uterus and the presence of the open canal of Nuck pull the ovary by the gubernaculum into the labium majus.

Before Hunter's ligament or the gubernaculum becomes adherent to the uterus, there is no substantial difference in the morphology of the two sexes, which may explain the failure of the ovary to descend. Deformity of genital development is undeniable; instances of combined gland or ovotestis (hermaphroditism) have occasionally been found in this type of hernia.

The hereditary influence on hernia is to be considered. Berger's report,[1] from an analysis of several thousand cases of hernia, reveals that more than one-third of the cases could be associated with heredity. Among the physiopathologic causes of hernia pregnancy stands first. Heinick[2] shows 79.9 per cent of the patients had borne children.

Inguinal ectopia of the ovary presents certain characteristics which are not found in simple ovarian hernia. In true ectopia the tube usually accompanies the ovary, which is explained by the intimate relationship between the genital gland and the upper part of Müller's duct. The cord is shorter and is tapered due to constriction, and like the testicle is classed as an extra-peritoneal organ, covered by a fold of the canal of Nuck. The peritoneal diverticulum is always present in these cases, partially obliterated or completely pervious. In both the ectopic ovary and simple hernia of the ovary, the ovary may undergo all possible alterations, inflammatory, degenerative or neoplastic. In the author's case the ovary was enlarged, the capsule was thickened and the ovary contained many retention cysts.

Ovarian hernia occurs most frequently in infants and children. Surgical measures should be instituted without delay, providing the nutritional index of the child will permit operation. The condition requires early recognition since, to save the ovary, surgical measures should be instituted before the circulation becomes altered and other catastrophes develop to necessitate removal. Wakeley[3] reported three infants (2, 6 and 7 months of age) who came to operation with good results; in each case the ovary and tube were replaced in the abdomen and permitted to develop normally.

CONCLUSIONS

1. Ovarian hernias are congenital. In children with ovarian hernia the ovary will appear at the inguinal region or labium majus as a small hard rounded swelling, movable and usually not tender unless there are circulatory changes in the herniated parts.

2. After puberty when a patient offers a history of the presence of a lump in the groin, whether stationary or not, and increase in the size and discomfort of the inguinal or labium majus mass at menstruation, hernia of the ovary should be carefully considered.

3. Surgery promptly instituted and carried out along the proper principles will save many ovaries. Delay and palliative measures may cause strangulation or pathologic changes.

4. It is obvious that ovulation in the ectopic ovary is useless for the purpose of reproduction and if the ectopia is bilateral, there is sterility. In simple hernia, even though bilateral, pregnancy can occur.

5. The Bassini type of operation offers the best results for the repair of hernia in the female. Local anesthesia, novocaine and ephedrine, is ideal for all cases except the infant and very young.

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SARCOMA OF THE OVARY

CASE REPORT

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Y. S., single, Japanese, aged 24, a housemaid, was seen January 25, 1937 because of a large firm rounded mass in the lower abdomen arising out of the pelvis.

She had been well all her life except for minor childhood illnesses, until at the age of 23 she noticed a mild non-radiating pain in her right lower abdomen. It came on insidiously and disappeared spontaneously after two days, although she continued with her housework. No lump was noted at that time and she did not consult a physician. Six months later a similar transient dull pain recurred. This time she felt a small hard lump low in the right side of the abdomen. Nine months thereafter this pain reappeared and she consulted her family physician for the first time. He ordered local application of heat and some oral medication for the relief of the pain. She was pain-free in four or five days. The mass was definitely larger at this time and the slow, progressive enlargement continued unabated. There were no more attacks of pain and she kept on with her work as housemaid, performing the usual tasks with but little discomfort. At no time was there any evidence of associated gastrointestinal, cardiorespiratory, or genitourinary distress.

The menses began at 14 and were entirely normal, with slight pain during the first twenty-four hours of the flow. The last period was fourteen days before examination, a normal five day flow. She denied coitus. The past and family histories were flatly negative.

The patient was a frail, slender, shy Japanese girl, in no apparent discomfort. Temperature was 99, pulse 92, respirations 20. The chest was thin, flat, and poorly muscled, the breasts small and undeveloped. Blood pressure was 124/76. The lower abdomen was asymmetrically enlarged, mostly to the right of the midline where a tumor, firm and hard, the size of a five month's pregnancy was felt. The right margin of the mass was round, the left borders smooth, though some indentation could be felt. The

upper edge of the tumor extended to the umbilicus while its lower limits extended deep into the right pelvis. No pulsation, bruit, funic souffle, fetal heart sounds, nor movements could be heard. On transillumination from the side of the tumor there was no indication of translucency. The upper pole of the tumor could be pushed about at will, but its lower pole was firmly fixed. No fluid wave was elicited and no other abnormal abdominal findings were noted.

On pelvic examination the introitus was very narrow, barely admitting one finger; the hymen was intact. The cervix was normal in appearance and consistency. The uterus was small, firm and in normal position. No abnormality was present in the left adnexal region, but the entire right fornix was occupied by a hard unyielding tumor anchored in the right adnexal pedicle and pushing the uterus slightly to the left of the midline. Only moderate tenderness was present and this only upon firm pressure upward in the right fornix by the examining hand. The tumor was not ballotable and there was no cystic feel to it. Rectal examination merely confirmed the findings on vaginal examination. Other physical findings were negative.

A flat x-ray plate of the pelvis showed no evidence of the calcific deposits of a teratoma. The blood count was normal except for a slight leucopenia. The urine, blood Wassermann and Kahn tests were negative.

A preoperative diagnosis of right ovarian cyst was made. This hypothesis was based on the following evidence:

1. Presence of a clearly palpable uterus entirely normal as to location, consistency, shape and size.

2. Presence of a large, firm, rounded tumor mass palpable distinctly to the right of the uterus, and rising up and out of the pelvic cavity.

3. Absence of clinical and roentgenologic signs of intra- or extra-uterine pregnancy.

4. Absence of symptoms or signs suggesting a process arising from the right kidney or intestinal tract. Due to the firmness of the

FIG. 1. Note homogeneous dispersion of the essential tumor cells and thin walled vascular spaces.

tumor, and its opacity on attempts at trans-illumination, it was thought that the tumor might be a solid or cystic teratoma, or an ovarian fibroma. A pedunculated myoma with a long stalk was also considered as a possibility.

Under cyclopropane anesthesia the abdomen was opened through a midline incision which was prolonged upward for an inch above and just to the left of the umbilicus. No free fluid was encountered in the peritoneal cavity. A hard, glistening white smooth oval tumor involving and apparently replacing the right ovary was encountered. It was non-adherent, and no raw surfaces were seen. The right tube was virtually obliterated by the tumor, which was pedunculated and was pushing a normal uterus to the left of the midline. The tumor was found to be twisted on its pedicle for three-fourths of a turn in a clockwise manner. It was untwisted, and the pedicle was severed and ligated. The raw surface was peritonealized and simple appendectomy was done. The uterus and left adnexa appeared normal, and were unmolested. The wound was closed in layers without drainage.

Convalescence was uneventful and the patient left the hospital on the fifteenth post-operative day in excellent general condition with primary union of the incision.

An x-ray photograph of the lungs taken on the eighteenth postoperative day showed no evidence of metastasis, and an Aschheim-Zondek test four months after operation was negative. More than a year postoperatively there had been no evidence of recurrence of the tumor.

The neoplasm weighed 2½ pounds immediately after removal and measured 14.2 by 12 by 9.5 cm. Its structure was peculiar. The capsule was very thin but entire throughout, except at the pedicle. The cut surface was quite homogeneous, and of a faint pinkish-white; it resembled the cut surface of testicle rather than ovary. Grossly, faint lobulations appeared, but fibrous trabeculae and stroma were almost altogether absent. Only very thick frozen sections were possible because of the high cellularity and this absence of stroma.

Microscopically, the stroma also was scarce, blood vessels and vascular sinuses were prominent (many of the latter formed only by a layer of endothelium). In the few areas of necrosis and in the scant connective tissue, lymphocytes appeared. The essential cell was a large, round, juicy one of very embryonal character. In some areas there was no order to the arrangement of these cells, in others, they were arranged in straight and twisted cords, the parallel ones sometimes simulating tubules. The essential cell had a rather scant cytoplasm but a very large nucleus, though not overly rich in chromatin. Atypical mitoses were very numerous, reflecting the rapid growth of the tumor.

We believed it to be a large round-cell sarcoma of the ovary, probably primary in the mesodermic portion of a solid teratoma. This last is speculative for it is obvious that serial sections on a 2½ pound tumor, in search of ectoderm and entoderm, are out of the question, but there is evidence that the representative cells of one of the blastomere layers in teratoma may proliferate at the expense of the other blastomere representatives till these latter are partially or wholly obliterated, probably from pressure atrophy. Frank[5] has pointed to the excessive proliferative capacity of the embryonal mesoderm and the fact that when a solid teratoma metastasizes, the metastases are
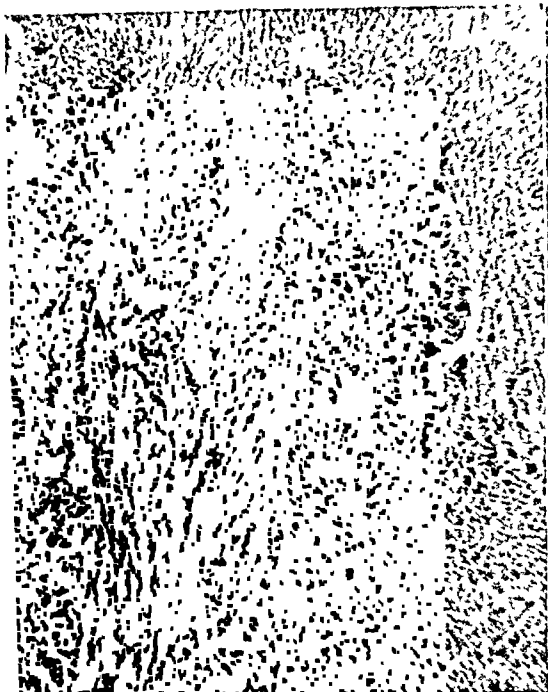
FIG. 2. Note occasional cord-like arrangement of tumor cells, simulating tubules.
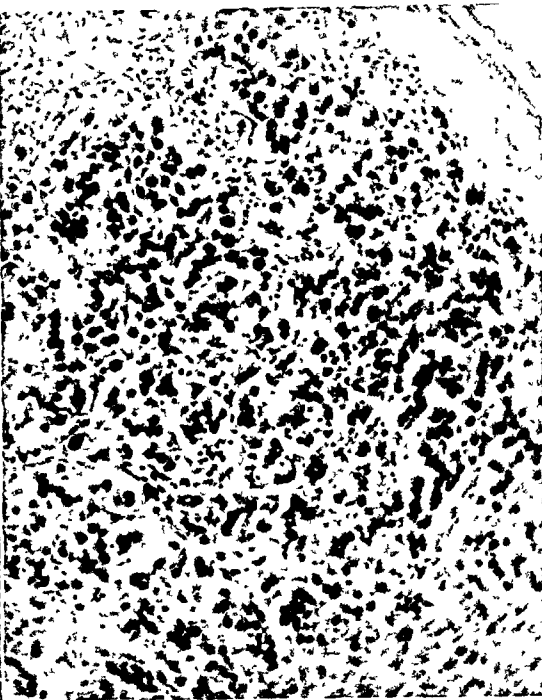
FIG. 3. Medium enlargement of an average field, showing the scattered lymphocytes.
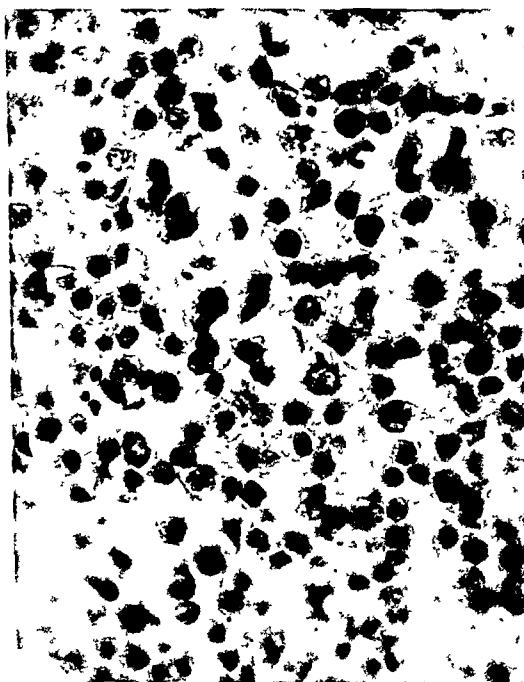
FIG. 4. High magnification to show the sparse cytoplasm, the empty intercellular spaces and varying quantity and quality of the nuclear chromatin. Mitoses are numerous.
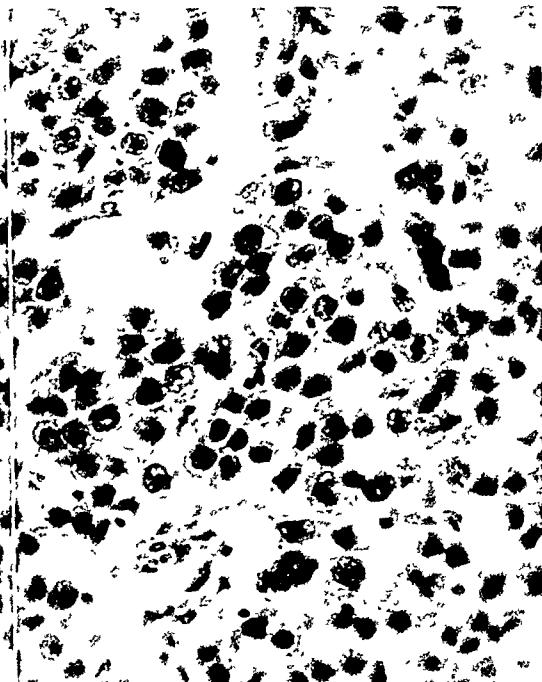
FIG. 5. High magnification, showing the character of the intrinsic cells, an arteriole and several mitoses.

composed mainly of mesoderm. This tumor seems to fit properly into the group of ovarian tumors known as *dysgerminomas* or indifferent cell tumors.[8]

COMMENT

An unusual type of ovarian tumor is presented. The patient was alive and well with no evidence of recurrence more than a year after unilateral oöphorectomy with removal of the tumorous ovary only. The hypothesis is advanced that the malignancy probably arose from a solid teratoma of the right ovary, though Gardner[7] points out that such malignant tumors are usually mixed. The subject of sarcomata of the ovary in general has been well covered by other writers including Scheffey,[12] Bland-Sutton,[3] and Kaufmann,[10] and is therefore not here dwelt upon. Careful examination of our patient at operation showed no ascites. This does not bear out Graves'[9] statement that ascites is present with all solid tumors of the ovary, nor that of Boyd[4] who states that ascites is invariably present with sarcoma of the ovary.

Opinion is divided as to whether one or both ovaries should be removed in this type of case. Anspach[1] takes the radical view in his textbook, although unilateral oöphorectomy only was done in his case reported by Scheffey.[12] Björkheim[2] has reported a case of malignant tumor of mesenchymal origin in a girl of 6 in whom unilateral oöphorectomy was done. That patient was well and attending school two years later.

It would seem advisable to have an immediate frozen section done on any solid tumor of the ovary, and to remove both ovaries should there be any suspicion of malignancy in the contralateral ovary. In any case of doubt, the Aschheim-Zondek test should be of value in ruling out recurrence of sarcoma arising from teratomas. A roentgenogram of the chest should be taken in every case of proved or suspected ovarian malignancy, preferably before operation.

Although a year's observation is too short to rule out any possibility of recurrence, the case is reported because of its unique features and the likelihood that we have a true cure. Conservatism with regard to the contralateral ovary may occasionally be justified in a case in which the ovarian sarcoma has a clearly defined capsule, is free from adhesions, if there is no fluid in the peritoneal cavity and if the ovary of the opposite side appears grossly entirely normal.

SUMMARY

A case of unusual ovarian tumor in a nullipara of 24 is presented. It is believed the tumor is a sarcoma, primary in a solid teratoma; it was very cellular and had very little stroma. Unilateral salpingo-oöphorectomy only was done without recurrence or metastases after eighteen months.

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MUCOUS ADENOCARCINOMA OF THE BLADDER*

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PRIMARY mucin containing adeno-carcinoma or colloid carcinoma of the bladder is a rarity. This case is reported as one quite typically characteristic of this infrequent grouping.

R. S., a white male, age 41, was admitted to the Jersey City Medical Center, January 23, 1936, complaining of intermittent hematuria for one month. This had started as a terminal bleeding of a few drops, with only occasional slight burning at the end of micturition. Apart from this, the patient was quite comfortable, experiencing no dysuria, urgency, difficulty, frequency or other untoward symptom. He daily pursued his occupation as mechanical engineer, without restriction. Gradually, the bleeding increased from a few terminal drops to a complete hematuria, with clots, mucus shreds and occasional finely granular calcareous material.

On admission to the hospital his red count was 4,470,000; 70 per cent hemoglobin (Tallquist); white count 5,350 with 66 per cent polymorphonuclear neutrophiles, 34 per cent lymphocytes. The Wassermann was negative. P.S.P. test: first specimen 52 per cent; second specimen 27 per cent. Non-protein nitrogen was 25 mg., sugar 104 mg. per cent. Other clinical laboratory findings and physical examinations were negative, as was the flat x-ray of the abdomen.

Cystoscopy revealed a small tumor at the apex of the vault of the bladder about 0.5 cm. in largest diameter, papillomatous and pedunculated, floating freely in the distended bladder. Its surface was irregular and appeared ulcerated and bleeding at one point. The remainder of the bladder mucosa appeared normal. A biopsy was taken and the tumor fulgurated. The patient was discharged the day following admission.

The biopsy at this time showed fragments of tissue composed of delicate villi lined with several layers of rather uniform transitional cells with conspicuous nuclei devoid of any demonstrable mitosis. The stroma was edematous, vascular and diffusely infiltrated with polymorphonuclear neutrophiles. The biopsy was considered inadequate to render a definite diagnosis. The possibility of papilloma was suggested.

Subsequent Course. The patient recovered, had no unusual urinary symptoms, voiding five to six times a day and only occasionally at night. Gross bleeding had ceased, but microscopic examination of the urine revealed usually some red cells and a considerable number of polymorphonuclear neutrophiles. About ten weeks after discharge from the hospital, a cystoscopic check-up was done. At this time, the tumor was again observed at the vault of the bladder. The neoplasm appeared definitely larger than on previous examination, measuring now approximately 2.5 cm. in diameter; it no longer appeared pedunculated, but infiltrating and ulcerated in its center which was irregular, covered with slough and calcareous deposits. The remainder of the bladder appeared inflamed and hyperemic. Resection of the tumor was advised.

The patient was readmitted to the hospital April 30 (three months after the first admission). On April 21, 1936, under spinal anesthesia a suprapubic cystotomy was done. The tumor mass was readily palpable through the vault of the bladder; the peritoneum stripped easily and the bladder mobilized. The peritoneum was opened accidentally, which permitted exploration of the abdominal cavity; no evidence of tumor was found therein. The peritoneum was then closed. The bladder was opened apart from the tumor site. A proliferating, soft, cauliflower-like growth about 2.5 by 2.5 cm. was presented on the anterior superior portion of the vault just slightly to the right of the midline. Whereas the surface appeared soft and necrotic, it definitely involved a greater portion of the thickness of the bladder wall. A smaller, firm nodular growth 0.5 cm. in diam-

eter was found about 1 cm. posterior to the larger tumor. With the electrocautery knife, the involved areas was removed; the resection

nary bladder" which measured 4.3 by 4 cm. in diameter. The peritoneal surface was slightly roughened by fibrous tags. The mucosa was

FIG. 1. Delicate villi lined with transitional cells; bladder wall invaded by neoplasm (low power).
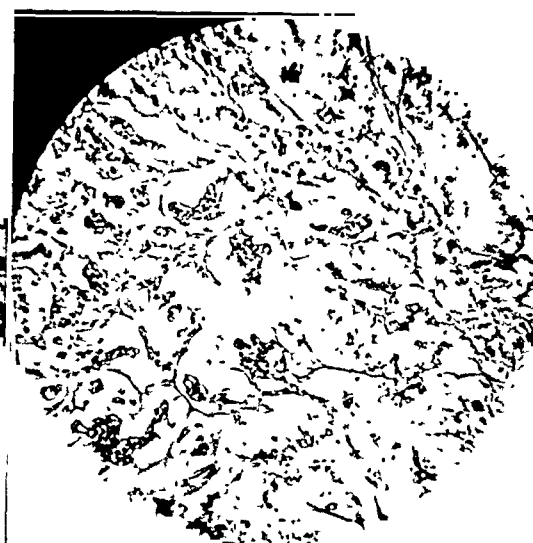
FIG. 2. Alveoli and cords chaotically arranged with conspicuous mucoid material (low power).

included a margin of 1 cm. of healthy bladder wall around the growth. The bladder was closed around a Pezzer catheter. Immediate convalescence was uneventful and the patient was dis-

FIG. 3. Cells proliferating to form lateral sacculation; cells are vacuolized and mucoid material conspicuous (high power).

charged in good condition on the twentieth day.

Pathologic Report. Gross description. The specimen, received in 10 per cent formalin, consisted of a membranous mass of tissue designated "anterior-superior portion of uri-

slightly trabeculated and its central portion presented a soft sessile mass measuring 2.3 cm. at its base. The central portion was ulcerated and slightly brownish. Section through the tumor revealed two-thirds of the musculature to be invaded by a translucent, granular tumor tissue which undermined the adjacent covering mucosa. One cm. posterior to the large tumor and connected with it by its base, was another similar mass which measured 0.4 cm. in diameter. The adjacent mucosa was trabeculated and markedly hyperemic.

Microscopic description. Sections showed delicate villi lined with six to eight layers of transitional epithelium limited by a definite membrana propria. The stroma was loosely arranged connective tissue with dilated blood vessels and a number of inflammatory cells. As the tumor was approached, the mucosa was abruptly interrupted by necrotic tissue, underlying which were pseudo- or glandular acini lined with low columnar epithelium often proliferating to form secondary alveoli. The nuclei were hyperchromatic and basally placed. The cytoplasm was faintly eosinophilic and often vacuolized. The glandular pattern was frequently lost and the cells were arranged in solid masses or cords; here the intracellular mucous material was more conspicuous as manifested by the presence of signet ring cells.

Even in this area, a tendency to glandular formation was noted. In places, the cellular element was absent and the scanty stroma supporting the preexisting glands was striking for the presence of mucous material. Mitotic figures were occasionally seen. The underlying two-thirds of the bladder musculature was invaded. Hemorrhagic and inflammatory areas were conspicuous throughout. The pathologic diagnosis was mucous carcinoma.

In view of the above report and with the realization that secondary invasion of the bladder by mucous adenocarcinoma from other organs was not uncommon, investigation of the gastrointestinal tract was performed. Proctoscopic examination and barium enema produced negative results.

Follow-up. Cystoscopy on June 8, six weeks postoperative, showed no evidence of the former or any new growth anywhere in the bladder. The wound was well healed, the patient looked and felt well and had resumed his usual vocation. Good health and freedom from symptoms continued until the middle of October, 1936, six months postoperative, when bleeding reappeared together with urgency and dysuria. Reëxamination at that time revealed an extensive tumor covering about 4 to 5 cm. of the vault of the bladder; it appeared ulcerated, covered with slough and deposits. Generalized infection was apparent elsewhere in the bladder; the inguinal nodes were enlarged, matted and formed a tumor of considerable size in the right groin.

From then on the patient's course was rapidly downhill. He became bed-ridden and two months later the right leg became painfully swollen from the ankle to groin, the inguinal lymph nodes increased in size to form a hard firm palpable tumor which was apparent well above the inguinal ligament—the tumor mass increased in size progressively. The patient died on January 28, 1937—just one year from the onset of the initial symptom. We were unable to obtain permission for an autopsy.

COMMENT

The histogenesis of this tumor is not known. It is extremely difficult to draw any conclusions from the few cases reported. The reviews of Campbell Begg[1] and Lee[2] adequately cover the etiology, symptomatology and incidence of this neoplasm.

This case presented several unusual features. Because of lack of microscopic confirmation of what seemed to be a clinical papilloma, it was deemed necessary to have the patient checked within ten weeks.

The rapid recurrence of the tumor in a resectable portion of the bladder made radical surgery imperative.

The final diagnosis of primary mucous adenocarcinoma of the bladder rests upon histologic findings, and negative exploratory laparotomy, proctoscopic examination and barium enema.

The innocent appearance of the tumor, the rarity of primary adenocarcinoma which accounts for merely $\frac{1}{2}$ of 1 per cent of all bladder tumors[3] and the inadequacy of our first biopsy, greatly contributed to a rapid and fatal termination.

CONCLUSIONS

1. Primary mucous adenocarcinoma of the urinary bladder is extremely rare.

2. Its tendency to early metastases and extreme resistance to radiation should warrant the classifying of the vast majority of cases as type III, Broders.

3. In this type of tumor, radical surgery in an accessible portion of the bladder is the only hope of prolonging life.

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NEW INSTRUMENTS

THE ALBEE-COMPER FRACTURE TABLE

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THE rapid growth of orthopedic, traumatic and reconstruction surgery during the last few decades has open operation or closed manipulative procedure upon any part of the body, particularly the extremities and the spinal
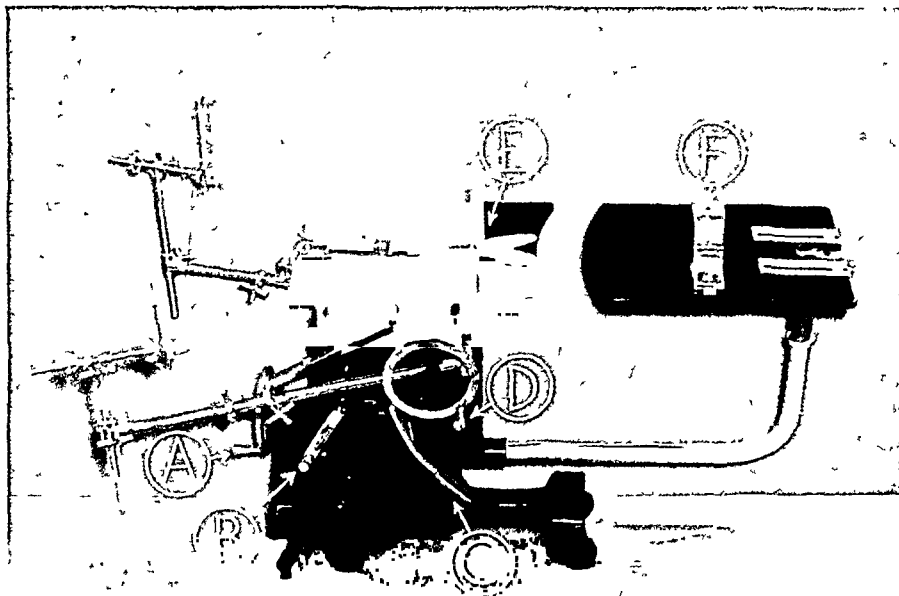
FIG. 1. The table in lateral tilt with head and buttocks section of the table top in position. The foot section is shown removed. Thus an assistant surgeon may be stationed between patient's legs in closer relation to the operating surgeon. Controls are indicated by A, Trendelenburg, B, lateral tilt, C, hydraulic handle for height adjustment of the entire table top and traction, D, control for lowering buttocks section of table top. For easy application of plaster hip spica cast, the head section of table slides back far enough to extend the cast to the axilla. Ample depth is provided so that there is no interference with placing the cast on an obese patient. Provides for overcoming posterior displacement of a short lower fragment of the femur. By placing a wood block or sandbag between the offending fragment and table top it may be levered into place by a turn or two of the wheel while internal fixation agent is applied. Similarly the rotation of the pelvis may be controlled by placing a block under the hip which may then be raised in relation to opposite hip by this movable portion of the table. Conveniently provides self-retaining retraction at lower edge of incision in open reduction work, the end of double rake retractor being hooked under draped edge of table top which, when lowered, automatically holds incision open. An important innovation is the application of control D in conjunction with the removable overhead frame as a crane at the end of the table, one of the principal uses of which is to provide positive gravity traction in Sayre suspension, as shown in Figure 2. E, perineal post and sacral rest are x-ray penetrable. F, body support used in lateral tilt.

brought very exacting demands upon the methods of holding the patient under traction both before, during and after column. The required demands are multitudinous in variety and most exacting in the precision necessary. Not the least of

these requirements are those developed in the treatment of various fractures and dislocations.

FIG. 2. Sayre suspension under control of crane for applying body cast, etc.

It has been increasingly realized that the innumerable variations of displacement of fracture fragments are due largely to unbalancing of muscular control by the solution of continuity of skeletal bones. Limb posture has been found to be of inestimable aid not only in reducing the fracture, but in the fixation of the fragments after reduction. This is due to mechanical causes, principally to the selection of a posture of neutral muscle pull, where the displacing influence of a muscle is offset by a posture relaxing it, while the traction is simultaneously applied.

It is becoming increasingly apparent that reconstruction and corrective surgery, made necessary by various infections, automobiles, sports or industrial accidents, is now being undertaken far more generally by the profession than heretofore. Much of this branch of surgery is necessarily of a major nature, where operative speed is of prime importance, but the

fracture tables at our disposal lack the advantages of a regular surgical operating table, and do not provide proper facilities
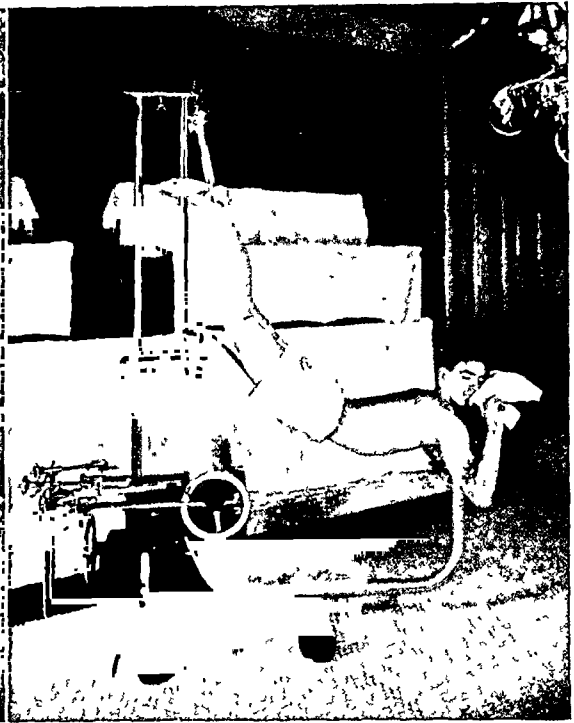
FIG. 3. Hyperextension treatment of fractures of spine (Davis position). A winch with canvas sling is placed in sockets on table top and when patient's legs are attached to overhead frame the table sections may be removed and hyperextension obtained by slackening canvas through control of winch. Precision control of desired posture and crane control D in vertical direction at foot of canvas enable sling to hug symphysis closely for accurate application of plaster jacket. Note that ample space is provided under abdominal region for management of obese patients in hyperextension and for x-ray or fluoroscopic examination in any plane without disturbing posture or immobilization of patient.

and adaptability for control of the patient; this, doubtless, is due to their being designed mainly for the reduction of fractures. Conversely, a regular surgical operating table lacks all means of applying traction during or after the operation.

An attempt to add traction apparatus to an operating table led to complication and limitation of general usefulness apart from creating blind spots in connection with x-ray and fluoroscopic examination. This experience, however, indicated the desirability of departing from the more con-
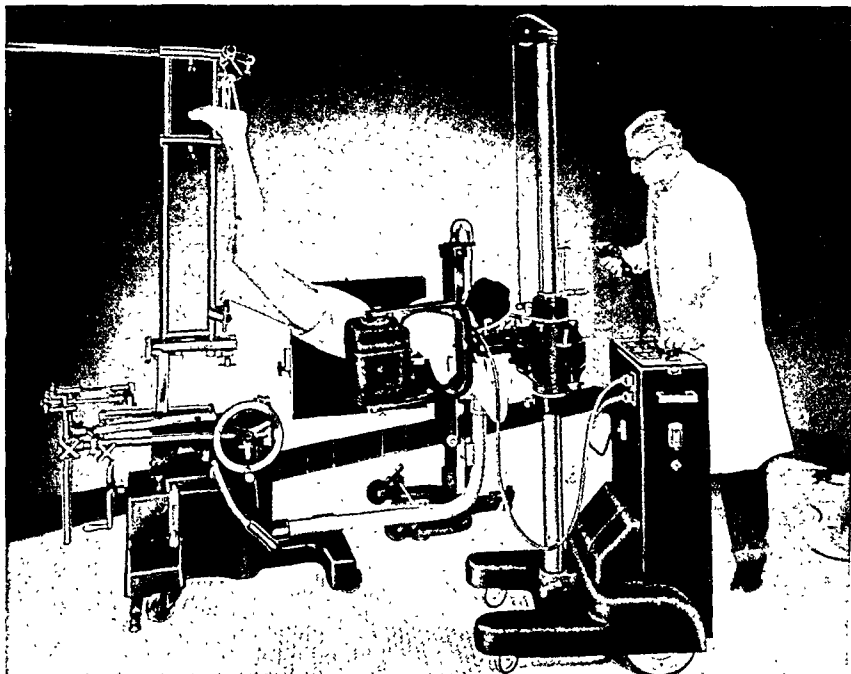
FIG. 4. Lateral radiography with mobile Potter-Bucky diaphragm. For antero-
posterior radiograph Bucky diaphragm may be placed at desired angle imme-
diately under patient.
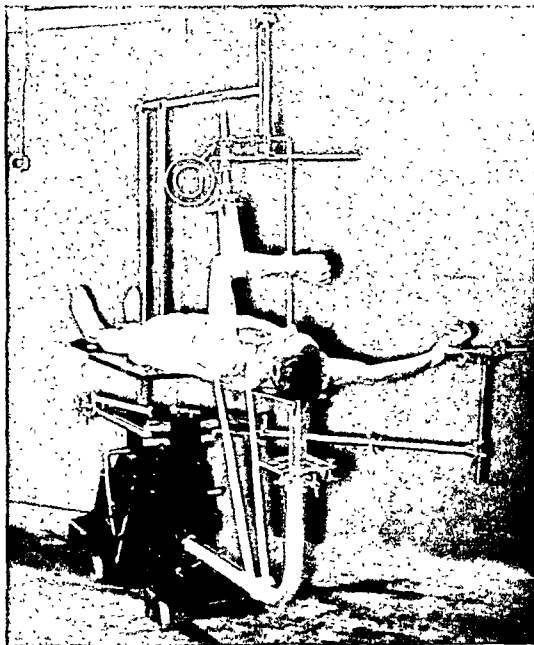
FIG. 5. Anterior elevated position. Turning
screw G applies traction, and anchorage to
structural tube of table provides very ade-
quate countertraction.
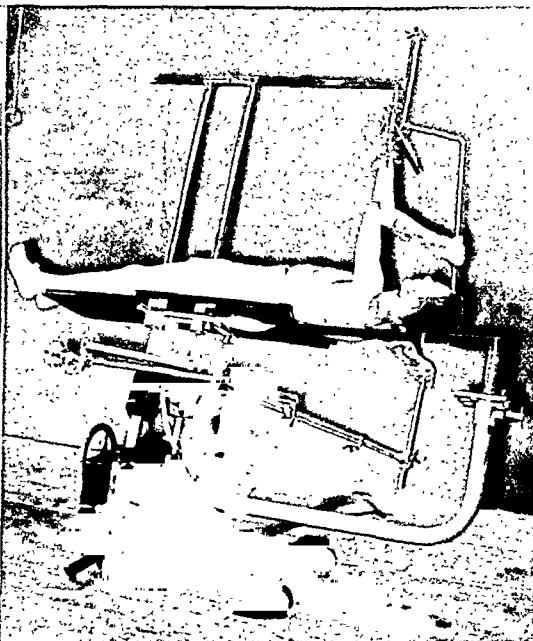
FIG. 6. "Salute" position for arthrodesis or other
operation of shoulder. Body support (not visi-
ble) is attached to table top at the waist; the
most favorable posture may then be obtained
by raising table to its maximum height and by
placing patient in lateral tilt. This is a great
advantage because when operating on any
table without lateral tilt, the surgeon must al-
most stand on his head in order to look upward
or laterally into the wound.

ventional standards of fracture table design by adopting those features of the surgical operating table which most usefully apply to traumatic and orthopedic surgery, and combining with them a simple and efficient apparatus for giving comprehensive orthopedic postures under traction, as well as providing for fluoroscopic and x-ray examination before, during and after the operation.

Based on this principle, therefore, with the efficient and ingenious aid of an engineer specializing in surgical equipment, Mr. Adrian Comper, I have designed a traction operating-table, which retains from the conventional surgical operating table:

1. Variable height range controlled by a hydraulic pump.

2. Lateral tilt, most advantageous for better positioning of the patient, particularly during hip and shoulder work (open fracture work, extra-articular fusion of the hip, arthrodesis of the shoulder), nailing or pegging for fracture of the hip, and particularly for lateral x-rays of such fractures.

3. Trendelenburg angle for spinal anesthesia or treatment of shock during the operation.

4. Compactness and maneuverability.

The combination of the first two exclusive features is of outstanding importance in enabling the surgeon to work with his patient at a height and angle best adapted to the occasion and his comfort.

The controlling mechanism is housed in a pedestal base as an entirely independent unit, resting on casters which may be locked at will. To this pedestal is attached another completely independent unit—a superstructure comprising the table top, traction apparatus, perineal post and sacral rest; consequently, when lateral tilt, for instance, is applied, no distortion or diminution of traction in relation to the patient occurs, and the perineal post and a conventional body support prevent any tendency of the patient to slide out of position on the table. In this superstructure are

embodied the following mechanical aids to the management of every desired orthopedic posture of both extremities, as well
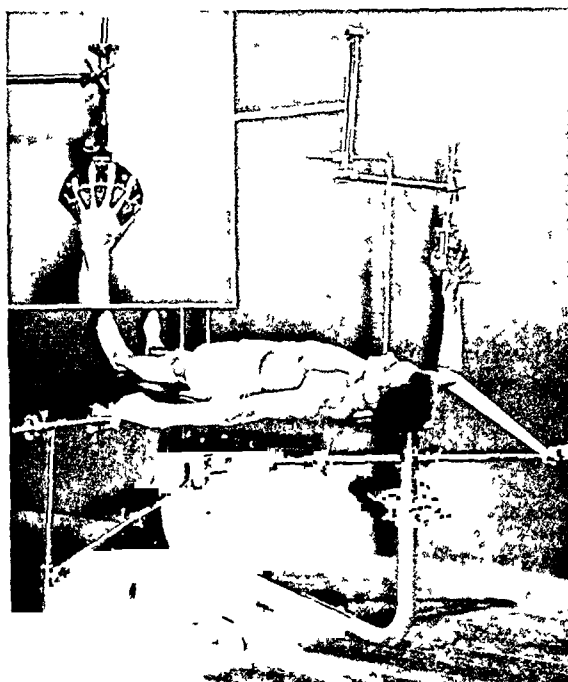
Fig. 7. Airplane position. Insert shows Weinberger hand and arm traction developed for Albee-Comper traction table.

as the spinal column, whether for open surgery or closed reduction.

The telescopic traction rods, universally adjustable for both upper and lower extremities, are of new design. They embrace the author's lateral adjustment of the upper end of these traction rods in accordance with the size of the pelvis, or intra-hip joint distance of each individual patient to insure their hinging immediately under the hip joints. The amount of traction thus remains constant throughout the excursion of the traction rods regardless of the degree of abduction or adduction applied. The importance of this device cannot be stressed too highly. The same lateral adjustment may also be used for special purposes such as reduction of fracture dislocation of the head of the femur, through the floor of the acetabulum. When the upper end of the traction rods are spread to their greatest width, the
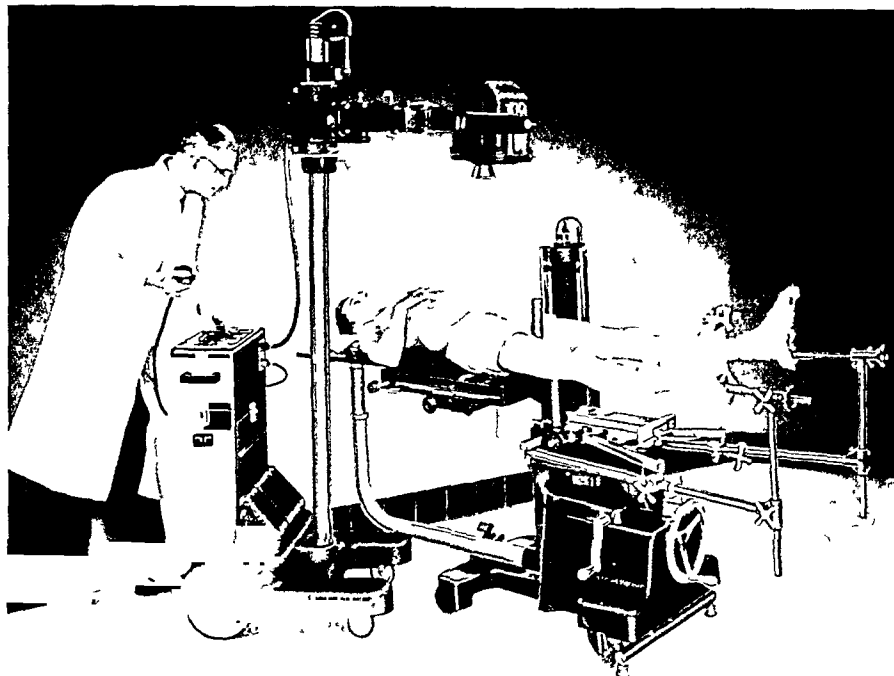
FIG. 8. Shows the advantage of the offset perineal post, making possible full pelvic
x-ray radiography. Note ample space under patient for insertion of Bucky diaphragm,
an application of film to upper third of both femora.
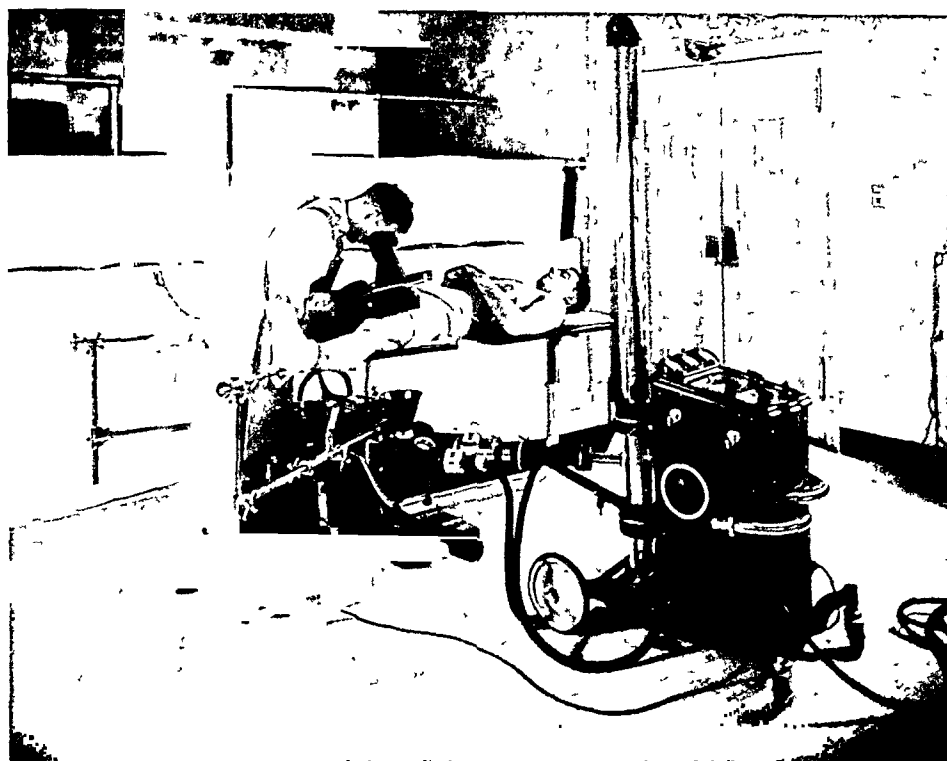
FIG. 9. Anteroposterior fluoroscopy or radiography of femoral shaft. Note uninterrupted
entrance between patient's legs for advantageous position of radiologist.

hinge point lies *outside* the hip joint; therefore, the amount of traction is automatically increased as abduction is increased, the

· *Radiography.* To allow accurate x-ray examinations before, during and after the operation, the perineal post—the datum
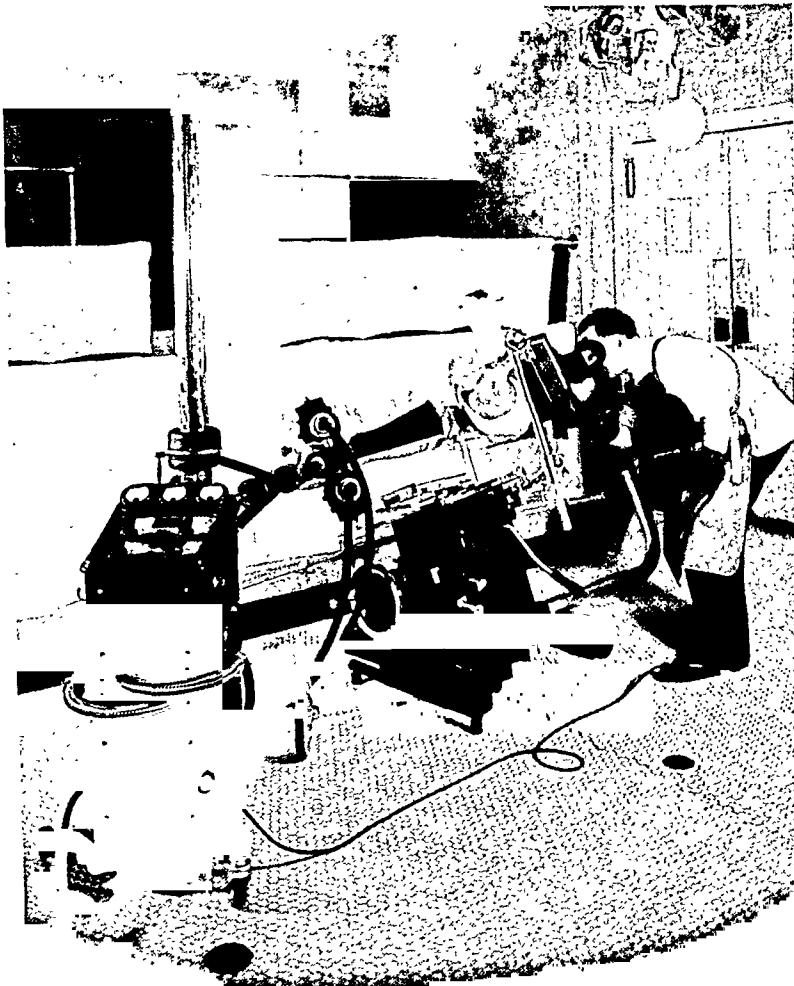
Fig. 10. Lateral fluoroscopy or radiography of neck of femur. Note superior posturing available by means of lateral tilt, which x-ray technician has found to be of the greatest help in lateral work on the hip.

maneuver thus providing an effective method of drawing the femoral head back into position through the floor of the acetabulum in the same direction it went in.

Management of Upper Extremities. Complete control of the upper extremity is obtained by substituting a narrow steel plate, readily withdrawn from the plaster cast, for the head section of the table. (Alternatively the hyperextension canvas sling may be used.) Traction arms, universally adjustable for *both* upper and lower extremities, are then swung into position and leg traction apparatus is removed and attached to overhead frame.

line of a fracture table—has been carefully positioned in relation to the pedestal base and a unique single tubular support has been adopted for carrying trunk section of table top. These two factors, together with an x-ray penetrable sacral rest and table top, now make possible complete radiographic or fluoroscopic survey of the entire spinal column and pelvic area by any shock proof mobile x-ray unit. The pedestal base slopes to a narrow top and traction arms have been kept free of protuberances for the purpose of providing unusually comprehensive anteroposterior and lateral views of the shaft of the femur

and tibia. The importance of x-ray check without removing the patient from the operating table cannot be too highly stressed under present day orthopedic and fracture conditions, and the unusually advantageous facilities for this purpose are self-evident from the accompanying illustrations.

It will thus be seen that the combination of many features of the surgical operating table with the conventional fracture table, and the development of additional mechanical innovations, has resulted in the consolidation of the two types of tables as one compact self-contained unit with unprecedented x-ray examination advantages.

In all fractures around the elbow the carrying angle of the arm, normally 16 degrees, must be maintained as nearly as possible. This is important to lend support to the elbow and to give strength for carrying burdens in the hand.

From—"Fractures" by Paul B. Magnuson, third edition (Lippincott).

A WOOD TUNNEL FOR SERIAL ROENTGENOGRAMS IN INTERNAL FIXATION OF THE HIP*

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INTERNAL fixation of intracapsular and intertrochanteric fractures of the femur ordinarily requires the taking of open end is braced with a strip of metal which extends upward on either side for about 1 inch to form an L. (Fig. 1.)
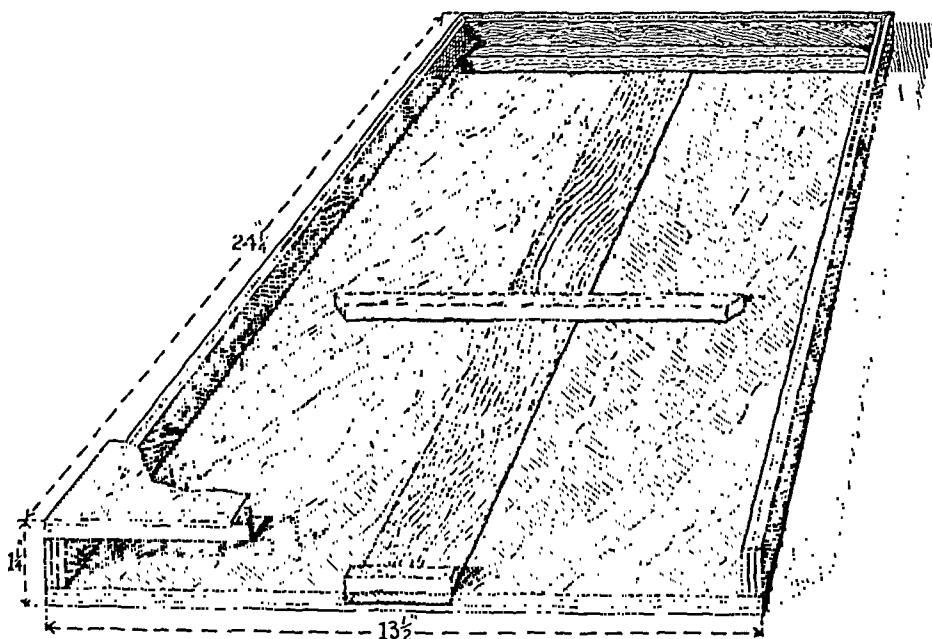
FIG. 1. Tunnel with upper section removed and film-holder in place. Note the metal brace at the open end and the channel through which the plate-holder slides.

several roentgenograms, which require in turn that the patient be shifted about on the operating table. The wood tunnel described has been devised to eliminate the necessity of these repeated changes of position.

The tunnel is a plywood box, open at one end, 1¼ inches high, 24½ inches long, and 13½ inches wide. The upper section at the In the middle of the bottom section (Fig. 1), extending its entire length, is a channel ⅛ inch deep and 2 inches wide, through which the plate-holder slides. The plate-holder is т-shaped. The long bar, which is 26 inches long, is the exact width and depth of the channel. The crosspiece, set at right angles to the long bar, is 1 inch high and 13 inches long. At the other end of

the plate-holder a similarly placed piece of wood, ½ inch high and the exact width of the holder, furnishes a grasp for the hand.

damp cloth. The plywood construction allows the rays to penetrate easily and casts no shadow on the film. The device is sturdy in itself, and the additional metal

PATIENT WITH TUNNEL IN PLACE. THE GREAT TROCHANTER IN LINE WITH THE MIDDLE OF THE TUNNEL

A

PLATE ON PLATE HOLDER READY TO BE INSERTED FROM THE UNAFFECTED SIDE

B

PLATE IN PLACE FOR A P VIEW

C

PLATE IN PLACE FOR LATERAL VIEW.

D
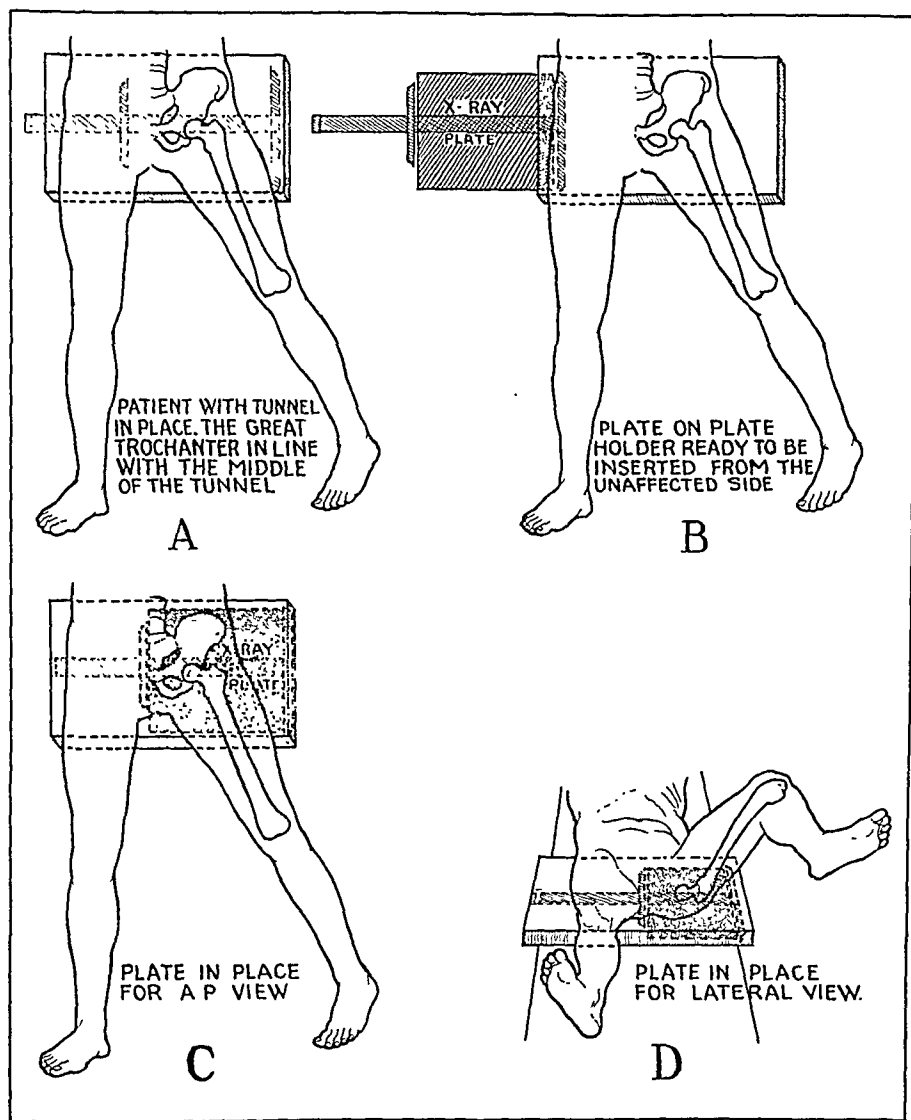
FIG. 2. Details of use for anteroposterior and lateral roentgenograms.

This plate-holder provides for a 12 by 14 inch cassette holding a 10 by 12 inch film. This is held in place by another crosspiece 6 inches wide. If films of different sizes are used, this crosspiece may be removed and the cassette held in place by adhesive.

The tunnel is shellacked, and blood and other stains are easily wiped off with a

brace makes its use possible with patients weighing as much as 250 pounds. The cost of material and making is less than five dollars.

When the tunnel is to be used, it is placed on the operating table, at the level of the hips, with the closed end toward the affected side. The patient lies on it, with the greater trochanter in line with the

middle of the tunnel. Pillows under the back, shoulders, and head bring the upper part of the body to the level of the tunnel.

pushed into the tunnel by way of the channel from the unaffected to the injured side, and as many pictures as may be

FIG. 3. Patient on table with tunnel under affected left hip.
FIG. 4. Plate-holder with cassette and film ready to be slipped into tunnel through channel.

Preparation and draping of the patient are conducted as usual. A film in a cassette is

necessary can be taken without moving the patient or disturbing the draperies.

A CONSERVATIVE TREATMENT FOR NONUNION

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THE splint design here illustrated is for the treatment of nonunion of the bones of the forearm. The same be adjusted to stay within the limits of comfort and safety.

The braces or supports for the distention
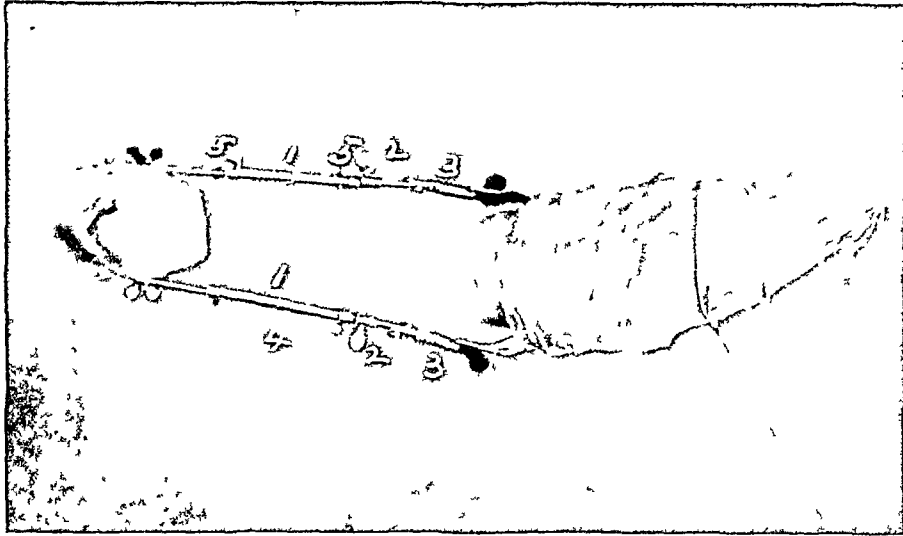
FIG. 1. 1, slidable distention mechanism 2, stop to prevent shortening of the forearm 3, rotating devices permit pronation and supination. 4, elastic bands giving continuing distention power. 5, bared hooks accommodating the elastic bands.

mechanism can be assembled on my splint designs for nonunion in fractures of the other major long bones. They conform to the new type of extension and of physiologic conservation incorporated in my splints for the immediate correction and treatment of fresh fracture deformities.[1]

The operation of the splint produces a continuing distention of the bone in its normal alignment, with opportunity for both flexion and rotation of the forearm. It proposes to secure by a continuing distention what cannot be accomplished by immediate extension of the injured member.

The distending power is confined solely to the forearm. It is not wasted over more extended areas. Thus less power is required to accomplish the same result.

The distending pressure is distributed along the length of the humerus and upon the carpal and the metacarpal bones. It can

are two unpadded casts. The cast upon the humerus has a soft pad over the vessels in the flexure of the elbow so that these vulnerable structures are protected absolutely from injurious pressure. The cast has a cap extending over the shoulder to prevent a drop that would occur otherwise upon the cylindrical arm. Metal plates with projecting ends are imbedded on the inner and outer sides of the cast at the distal ends. These plates are so placed in the cast that terminal holes in the plates come opposite the axis of the elbow joint. An unpadded cast is moulded to the hand covering the carpal and the metacarpal bones. The fingers are left bare to permit function. Metal plates with projecting screws are imbedded on the dorsal and palmar aspects of the cast.

The distention mechanism (1) is composed of two splint arms on each side of the limb. These splint arms have appropriate

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sleeves, each for the engagment of its mate. They can thus slide upon each other. Hooks on these sleeves are for the attachment of rubber bands for the continuing distention. A stop (2) on the upper splint arm prevents shortening of the forearm beyond the adjusted position.

The rotating devices (3) are separate units attachable to the proximate ends of the upper splint arms. They are not needed in the treatment of major long bones other than those of the forearm. The distention rotation mechanism is attached to the arm and hand casts as shown in Figure 1.

With manual traction the forearm is stretched and this degree of distention is held by adjusting the stops. Additional distention power to the limit of comfort is secured by stretching rubber bands (4) between the hooks (5) upon the splint arms.

The patient is encouraged to use the limb. It will be noted that the splint arms substitute for the broken bones. They give a stability in normal alignment and permit the exercise of more power and a greater degree of usefulness of the arm. Controlled function of the arm gives greater encouragement to nutrition and hence to bone regeneration.

If with a satisfactory approximation of the fragments bone union does not occur after a full probationary period, one can consider the advisability of plunging a trocar and cannula between the fragments. With the trocar or with a rasp the bone ends can be denuded and the intervening fibrous tissue broken up.

I propose this method of treatment patiently pursued for a lengthy period as a substitute for the more radical operative procedures with their attendant uncertainties and dangers.

REFERENCE

1. Masland. *M. Rec.*, Sept. 19, 1934.

BOOKSHELF BROWSING

HUGO OF LUCCA AND THE SCHOOL AT BOLOGNA

INCUNABULA MEDICA IV

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SAN FRANCISCO, CALIFORNIA

WHEN, because of war with its later discrimination and persecution of the defeated, it became necessary for the studious minded at Salerno to seek refuge elsewhere, there were open to them three avenues of refuge. First of these was the University at Montpellier in France, at that time in the process of growing, not yet within reach of its great fame which was to come later. Secondly, there was the University at Paris, in about the same situation as Montpellier, yet a bit closer to its zenith than the former. This later fame was to result from the very conditions causing the eclipse of Salerno. Several brilliant minds of the century came to the Paris faculty who would have gone to Salerno otherwise.

The studious intellectual mind, more particularly if of a philosophical bent, requires for working a scene of peace and tranquility that there may be opportunity for deep speculation with freedom from such environmental factors as discipline, dictation, regimentation, persecution and strife. Apparently conditions have altered little from those days of 700 years ago, since today in our own times we see an exodus of just such personalities from one part of the world and a migration to another, seeking exactly that same haven of peace and solace. One such brilliant personality at Salerno, seeking a new haven where he could work in his own manner, is interesting since he furnished a link in the chain of the dissemination of medical knowledge. Probably because he was a loyal and patriotic Italian, and perhaps because of the much shorter journey necessary from Salerno, this individual chose not Paris or Montpellier, but the third of the available possibilities, the University at Bologna.

The part which it is possible for a single great mind or a great teacher to play in the field of art and science has been repeatedly demonstrated throughout history. In the days of the early centuries, students flocked from all parts of the known world to a great teacher or a great master. Where they gathered in sufficiently large numbers and the faculty was of outstanding caliber, a center of teaching grew, or a university, and it was in such manner that Bologna began and grew.

One particular individual who was to be instrumental in helping its growth and fame, was Hugo, who, because he came from the Italian town of Lucca, was called Hugo of Lucca. As a surgeon he was not

without training or experience, having been associated with those two great names of the twelfth century, Roland of Parma and Roger of Salerno for many years. Bold daring surgeons were these latter and neither the teachings of Constantin, nor the school of Salerno could influence them. Their work was of such outstanding brilliance and accomplishment, that they could stand entirely alone and unaided without any reflected prestige from any university, even the famed Salerno.

Among their many contributions they described syphilitic or venereal lesions almost 300 years before Columbus returned from America (another nail in the canard that syphilis had its origin among the Indians in America and was brought back into Europe by Columbus' sailors). They described the lesions of cancer, particularly superficial skin cancers, quite accurately. Internal cancers more often they confused with syphilis, yet that is done even today.

The use of seaweed, the eating of it or decoctions of it in the treatment of swellings of the neck, such as in goiter or tuberculous adenitis was advocated by both. In other words they "Iugolized" their goiter patients as we do today, except that manufacturing facilities not being available, they obtained their drugs in the crude stage and so used them. Laudatory biographies of both these brilliant personalities have been written by many students of medical history and are available to those who wish to read more about either of them.

The consensus of opinion among students of medical history is that Hugo of Lucca surpassed even his teachers, because they followed more or less blindly, dogmatically and unquestionably that which had been set down in manuscript by their illustrious predecessors, whereas he possessed a faculty of questioning everything, of applying a certain practicality to everything, every formula or procedure, simplifying it, and discarding that which he found not true in actual practice.

In the practice of medicine there have been from time to time such men. They fail to follow in the groove of custom because their own judgment tells them not to. There have been and there will always be great practical teachers of medicine who devote their entire time to study and teaching and practice, like Hugo. Having little time to write or express themselves on paper, their passing from the scene is usually marked by a sparseness of available writings. Posterity suffers thereby, but these men are usually so practical-minded that the wide publication of one of their methods, which to their own thinking is of such utter simplicity as to be almost ridiculous, is distasteful, savors of personal aggrandizement, and they look upon it with disdain. So it was with Hugo. Exceedingly small is the amount of his writings which can be authenticated, though he lived and practiced until he was about a hundred years old.

One fortunate circumstance lessened the possibility that all his work would be lost. He had several sons, four of whom decided to make medicine their field. One, Theodorich, called Theodorich Borgognoni, he was particularly attached to and made him his intimate associate, assistant and confidant.

Because of a feeling that the teachings of his father should be made known to the scientific world, Theodorich took it upon himself to gather together and publish several tracts, the most famous of all being a "Cyrurgia." Two hundred years or so later, in 1498 and 1499, these were gathered into print and published in Latin in Venice and in French in Lyons. These two items today form very rare incunabula, difficult to obtain at almost any price.

Due to the constant interchange of ideas, practice, and writings of father and son, Hugo and Theodorich are usually spoken of together and their writings are considered jointly.

In order to understand why Hugo of Lucca is considered as one of the greatest of his contemporaries, an examination into some of his surgical ideas and procedures is of help.

First, and perhaps most simple, were his ideas as to the etiology of headaches. He distinguished them as being either of brain origin or derived from a state of disturbed digestion. He stated, "Those found to be due to some cause within the skull or in the brain itself, such as might be caused by the pressure of a tumor, will be found to be constant and continuous, without any relief whatsoever, even for an instant, while those arising from a disturbed state of digestion will be found to have intervals of complete freedom, then very intense and severe intervals dependent entirely upon the state of the stomach."

Because war and civil combat were the order of the day, the most common injuries were those occurring from bludgeoning of the skull by heavy weapons wielded with great force and producing all types and degrees of skull fracture. It was obviously necessary that the surgeon of the time be most experienced and skilled in the handling of such a type of injury.

The next most common type of injury was that caused by arrows. These wounds called for no end of ingenuity on the part of the surgeon. The making of arrow heads invariably exhibited an unusual degree of malicious invention. Some were made in such a manner that when they penetrated into the flesh they could not be withdrawn without extensive incision into and damage of the tissues for a considerable surrounding area. In such cases Hugo evolved a technique entirely his own. He made no attempt to extract the arrow immediately upon penetration, endeavoring first to disinfect the wound with hot wine applications. Then in a few days as the muscular rigidity relaxed, it was easier to extract the arrow head, often without any additional incisions whatever. Theodorich, in describing this method of his father's relates, "Once I saw two strong men make efforts to extract an arrow from the body of one who had been so injured. Despite their great strength they were unsuccessful and the injured one was brought to my master, Hugo. By the application of his usual remedies in a few days the arrow withdrew almost of itself."

When it is considered that every ingenious expedient that the brain of man could devise was used to tip these arrow heads, such as dipping them for long periods in poisonous concoctions or smearing them with known poisons, anything that would increase the deadliness of the weapon, then it is not hard to visualize that the problem of wound infection and its handling was no minor one.

Here also he had his own particular technique. All wounds were dressed with wine, and he emphasized that it was important to use the best possible wine that could be obtained. In the hands of an honest individual this admonition was probably very wise. It has been related that contemporaries and followers of Hugo carried out this admonition with rather shady results, setting aside the good wine for personal consumption and using for the wound any other that was at hand. Human behavior down through the centuries has probably been no better and no worse.

Hugo described the penetrating wounds of the chest caused by arrows, the resultant empyemas, the complications of rib fractures, the lung abscess and lung gangrene which often followed. He reached a peak of surgical accomplishment by performing a complete lobectomy, although involuntarily. In describing this particular case, Master Hugo relates:

"There was a nobleman living in Bologna, named Domicellus whom I was called upon to treat. When I arrived within the walls of his house the stench of rotting flesh was of such intensity that I thought it almost unbearable. On approaching his bedside, I noted that his once huge frame had shrunken to an almost unbelievable degree, both from the nature of his wound and from the wracking of his body with pain. As I stood there, he coughed up quantities of the most foul smelling substance in which were great pieces of substance and when I examined his wound which was an enormous one of his chest,

I could see the putrefying substance of his lung and I noted that that which he coughed up was exactly the same as I could see in his wound. As I proceeded to dress his wound according to my usual procedure I thrust my hand into the wound and on withdrawing it found that it contained such a huge portion of lung substance that I became alarmed. However, I was able by my method to heal this man completely even though a part of the lung was lost."

In the treatment of wounds Hugo very often differed from the strongly entrenched medical authorities, but he was very careful always to mention wherein he differed and why. He casually mentioned that he was quite aware that Galen or Avicenna recommended some other procedure or strongly advised against the very steps he was using, but then he cited his reasons, and, as though to clinch the matter, gave names and places where he cured so and so by this very method. Nothing could be more convincing.

His son, Theodorich, who was working with him constantly and absorbing as much of his father's wisdom as he could, speaks of the treatment of these wounds thusly: "When a white pap-like fluid runs out of the wound, then things are not too well, and when a very thin serous-like fluid comes out then there is great danger, but one must not despair wholly because I personally have healed such men following the treatment advocated by my father and I have seen a great many who showed the above mentioned signs healed by him."

The third type of traumatic injury most commonly met with was that caused by the sword. These wounds varied from minor lacerations to cuts of extreme degree. If one has noticed in the museums the varying type of weapon which came under the heading of sword, one can well visualize the extent of injury which could be inflicted by a husky expert wielding such an instrument, particularly those enormous two handed and double edge blades.

Hugo describes the wound produced by just such a two handed affair wielded by a giant of a man which caught his opponent across one side of the skull. Not only did it trephine him, but it accomplished a partial decerebration much the same as that achieved by our research men with great pains. The victim did not die, much to the astonishment of Hugo himself and the physicians of Salerno. The latter had refused to undertake his treatment, forcing him to journey to Bologna to Hugo.

In Hugo's treatment of this case he stressed the necessity for the utmost cleanliness in and about the wound, the careful shaving of the surrounding skull, the wisdom of avoiding the probing of such a wound needlessly or too many times. The actual treatment was to fill the cavity with a consecrated oil, "olei consecrationi," then to apply a pressure tamponade, snug and firm but free from any unnecessary force. Theodorich, in describing this particular case states, "One ventricle of the man's head was totally empty of brain substance, nevertheless he was healed by my father Hugo who restored the brain substance by flesh (this presumably means connective tissue) and as it was the cellule of memory I noted that he was very astonished that the man retained his memory as before."

This entire description one can accept with the proverbial grain of salt.

Theodorich stressed the fact that these types of wounds when treated by his father from the very beginning and dressed only by himself, invariably did well. This leaves, one presumes, a loophole for Hugo, because his cases invariably came from a distance and had been treated in some form or another by other surgeons in other localities. The lack of success, when it did occur, could thus very conveniently be shifted back to the original physician. The same type of alibi is used today with not the slightest deviation in method.

As can well be imagined, Hugo's success with such outstanding cases as the man with the gangrene of the lung and the other with the loss of brain substance brought

him a fame far beyond the geographical borders of his town and his country. His practice grew enormously and he was called everywhere in consultation.

tions of Avicenna. It seems that Avicenna advocated attempting to sew the structures together whereas according to Hugo perfect approximation of the structures by

PROLOGVS

CHIRVRGIA EDITA ET COMPI
lata ab excell. domino fratre Theo=
dorico episcopo Ceruiensi ordi=
nis prædicatorum.

Capitulum proæmiale.

VENERABILI patri & amico charissimo egregio viro dño .A. dei gratia episcopo Valetiñ. frater Theodoricus eiusdem patientia Botoniañ. ecclesiæ minister indignus: opus diuturni affectatum. Dudu pater charissime, Romæ pariter existentes, me vestrum tunc temporis capellanum et pœnitentiariū domini Papæ affectuose rogastis: vt vobis quædam artis medicinæ chirurgiæ, scilicet occulta & implicita, & ab antiquis imperfecte dicta, librum super hoc faciens secūdum medicationem domini Hugonis de Luca uiri in prædicta scientia peritissimi, aperte describerem: & breuiter explicarem. Ego vero volens in parte, & si non in toto satisfacere vobis vestris, librum ecce edidi quem eo tempore, sicut nostis, imperfectū & incorrectum more impatiens vobiscum in Hispaniam deportastis Rogantes, et postmodum per nuncios crebris literis expetentes, vt vobis eundem librū suppletis defectibus trāsmitterem emendatum: diu distuli. Sed ecce iam appropinquante senio, Chri...

Vero modico valde tempore fui cū domino Hugone prædicto, neque videre, neque comprehendere, neque discere ad plenum potui experiissimas curas suas: ideo in parte ista imperfectum meum ex propria experientia, & antiquorū curabo perficere, Galieni maxime, quem a prædicto viro eximio in nullo nouimus discordare. Vt autem ad librum istum acessus facilior habeatur, quid sit chirurgia, vnde dicatur, quales operatores habere debeat, necnon quæ sit ipsorum intentio, & in quot diuidatur species, breuiter denotabo.

Chirurgia igitur est operatio manualis in corporeanimalis ad sanitatē tendens. Vel sic. Chirurgia est vltimum instrumentū medicinæ. Tria enim sunt instrumenta medicinæ, quibus mediantibus, morborum causis valet medicus subuenire, scilicet diæta, pono, & chirurgia. Diæta est primum instrumentum: & est melius, sicut testatur Gal. in cōme to regiminis acutorum dicēs. Si possumus hominem curare cum diæta non curemus eum cum aliqua potiōe. Et Damas. in Aphoris. Si poteris hominem cura re per diætam, prosperam inueniens. hoc aūt instrumentum siue regimen exigitur contra causam, & facit digestionem. Secundum regimen seu instrumē tum est potio de qua idem Gal. Si cum potione infirmitatem possumus remouere, ad chirurgiam recurrendum non est. Si autem per duo prædicta regimina seu instrumenta remoueri non poterit: remoueatur beneficio tertii regiminis: videlicet chirurgiæ. Dicitur autem chirurgia a cheir quod est manus, et

ergein quod est agere: inde chirurgia, quasi manualis operatio.
Oportet operatores chirurgiæ, vt dicit Halyab. in commento, frequentare loca: vbi periti chirurgi operentur, & eorum operationes diligenter attendere, et memoriæ commendare. Oportet etiam vt non sint temerarii, neque audaces, sed sint prouidi, suaues, & cauti: vt cum maxima deliberatione & suauitate omnimoda, qua potuerint, operentur: & maxime circa cerebrum, panniculos, membra neruosa, & loca alia tumorosa. Et quia in libris comprehendi non possunt omnia quæ necessaria sunt ad artem: & frequenter multa operatori occurrunt, quæ non potuerunt de facili prouideri: oportet medicum ingeniosum esse. Damas. enim multum cōmendat in medico ingenium naturale, dicit enim. Ingenium medici naturale adiuuat artem, & naturam regentem. contrario vero contrarium. Oportet etiam esse litteratos, alias, et si interdū experimentis iuuentur, frequenter tamen in errorem incident, & labyrinthum. vix enim aliquem sine literis puto posse comprehendere chirurgiā. Refert enim Almāsor, q operatores huius artis pro maiori parte idiotæ, rudes, & stolidi sunt & causa stoliditatis eorū ægritudines in hominibus pessimæ generantur. Plerunque etiam cum non operantur sub certa radice, neq rationabiliter propter ipsorum imperitiam homines occiduntur. Causas etiam, & nomina infirmitatum quas asserunt se sanare, penitus non agnoscunt.
Intentio operationis chirurgorum est circa tria, scilicet circa coniunctionem solutorum, circa separationem coniunctorum præter naturam, & circa exurationem superflui.
Species chirurgiæ, sicut testatur Ioānitius, duæ sūt. Dicit enim, chirurgia est duplex: aut i carne aut in of se. Et secundum contrarietatem membrorum subiectorum: nam alia fit in membris mollibus, vt in carne, neruis, venis, & similibus. Alia fit in membris duris, vt in ossibus, & consimilibus quæ sicut differunt in contrarietate qualitatum, scilicet mollicie. & duriciæ: sic et in omni operatione chirurgica. Chirurgia quæ fit in membris mollibus, subdiuiditur tripliciter: aut fit in carne tantū, quæ scarificatio, vel incisio, seu futura, aut incisio nuncupatur. Aut fit in uenis nō pulsantibus: aut in pulsantibus: & dicitur phlebotomia siue sectio. Aut fit in neruis secūdū latū, et tūc incisio notatur. Aut secūdū longū, & tunc punctura dicitur vel scissura. In diaphragmate tā & panniculis, nominatur ruptura. Chirurgia vero quæ fit in membris duris, duplex est: aut enim fit in ossibus duris, & dicitur restauratio siue consolidatio fractorum aut fit in ossibus dislocatis a loco proprio, & appellatur coniunctio disiunctorum.
Vt autem quod vnusquisque voluerit, valeat facilius inuenire diuidemus in quatuor particulas librum istum ordinate per capitula partes singulas distinguendo ita tamen q in prima parte huius libri non in speciali, sed sub quadam intendimus generalitate tractare.
Diuiditur autem liber iste in quatuor partes. In prima parte agitur vniuersaliter de vulneribus, vice ribus, de fluxu sanguinis, & medicinis ipsorum, de iu dicēs mortis, de vulneribus neruorum, cura apostematis calidi, atque spasmi. In secunda parte tractatur

FIG. 1. Page from Hugo and Theodorich's "Cyrurgia."

There were apparently many deep sword wounds of the neck, often involving the great vessels, the direct sequel of attempts to decapitate the opponent. Here Hugo's therapy was unique and individualistic. According to his custom, he humbly apologizes for his technique, stating that he knows he is contrary to the recommenda-

pressures and pads was all that was necessary.

In the treatment of ordinary lacerations or cuts he was considered a master. His approximation of edges was so perfect, that his son often stated, "My master Hugo heals and consolidates all wounds with wine and compresses, making a convenient and

artistic dressing which he knows how to do so well, thus producing the most beautiful cicatrices without the use of either sutures or salves. No ragged or stringy cicatrix ever remained from the wound or abscess of a man he had treated himself. In conformity with this I have never seen a single broken or cut blood vessel which he was not able to close by the use of such a dressing."

Apparently the greater proportion of these civil and war traumatisms were not superficial, but penetrated deeply as Hugo describes. He stresses the painful symptoms which accompanied the enclosure of a cut nerve ending in a scar. However, he did not advocate the suturing of the cut ends of nerves even though they were easily visible and it could be done very readily. He claimed that his method of treatment by pressure and approximation was efficacious in all wounds whether nerves or blood vessels were involved or not.

He recommended the most exact conjunction of the surrounding tissue, this approximation to be maintained by little "cushions" (compresses?) which acted also to keep the wound dry. "When it became absolutely necessary the sides of a wound might be sewed without, however, touching the nerve itself. . . . The ends of a disunited nerve shall be brought together by pressure upon the surrounding tissue only." He recommended massage with a special oil of his own formula if the nerve ever did get entangled in the scar and was painful.

In his septic wounds, he threw overboard the theory which was then and later called that of "laudable pus," and used little cushions soaked in warm wine to clean them, then dried them thoroughly. Sometimes he used an "attractive smelling cleansing desiccant applied by a suitable dressing which the master could make very well." Hugo constantly admonished that wounds could not be healed by either incantation or magic powders nor by cauterization with heated stones as was done in many places.

He apparently recognized "septicemia" secondary to infected wounds, stating that

when the virus escaped from the wound into the interior, then a warm decoction of roses and various other substances was to

20 De apoftemate mamillarum, & virgæ.
21 De fclirofi.
22 De tumore.
23 De puftulis albis quafi puncta, quæ apparēt fub nafo: & fuper pomis maxillarum.
24 De fcroffulis.
25 De glandulis, & nodis.
26 De teftudine.
27 De nodis fiue teftudinibus contingentibus in capite: & dicuntur cornua.
28 De nodis fiue lupus quæ nafcuntur in palpebris oculorum.
29 De bubone.
30 De bocio.
31 De napta.
32 De inflatiōe fiue pinguedine quæ apparet in mamillis quorundam hominum.
33 De hydropifi.
34 De omni eminentia et ruptura: quæ acciditin fiphac ventris.
35 De hernia inteftinali.
36 De hernia aquofa, ventofa, et carnofa.
37 De apoftematibus tefticulorum.
38 De verrucis, & porris accidentibus in virga, uel in alia parte corporis, et de clauis, et formicis.
39 De clauis fiue callis.
40 De cura formicæ cum ferro.
41 De hæmorrhoidibus.
42 De fiftulis in ano.
43 De ficu facto in ano.
44 De lapide veficæ & renum.
45 De cauterijs & iuuamento ipforum: & quomodo fint in quibufdam ægritudinibus faciēda.
46 De combuftione ignis, aquæ, & olei: vel aliquo huiufmodi.
47 De fcabie, & pruritu.
48 De puftulis faciei.
49 De malo mortuo.
50 De impetigine, & ferpigine.
51 De gutta rofacea.
52 De pannaritio.
53 De morphea.
54 De lentiginibus, panno, rugis, liuore, nigredine.
55 De lepra.
56 De tuberibus.

FIG. 2. A list of varying subjects published by Hugo and Theodorich, evidence of their versatility.

be given internally. But the wound was to be continuously treated according to his method despite this escape of virus, and he remarked that if it had been treated correctly the virus could not have escaped into the interior.

That he was able, one can hardly doubt. The description of his method of reduction of fractures by first immersing the patient in a hot bath thereby relaxing all involved structures, then slowly and carefully teas-

ing or coaxing the fragments into alignment, is brilliant. In fractures of ribs he used gentle massage and compression much in the manner in which we do artificial respiration, pressing inwardly where necessary with one hand and massaging lightly somewhere near with the other until he had obtained that degree of approximation of fragments he desired. In similar manner he reduced dislocations.

A description by his son of such a fracture reduction is as follows, "The master Sir Hugo, led the patient into a warm bath where he was instructed to lie down. Dipping his hands into first turpentine, then honey and then pitch, Hugo laid his hands on the place where there was the weakness, pressed inwardly then lifted his hands suddenly. This procedure was repeated quite often until the ribs were brought back into a normal position after which a dressing of pitch and tar was made to hold this position." In the matter of splints for fractures, he did not recommend them or use them much. He preferred thickened dressings which could be moulded by hand rather than those made of "timbers" as he described them.

Compound fractures he described in detail. Their treatment was to consist of the application of both his methods. The first, reduction of the fracture itself, the second, his usual method of treatment of any and all wounds.

That he was humane in his work can be judged by his descriptions of his "spongia soporifera" to induce narcosis in those patients where pain could not be avoided. Although there is much evidence that there were many varied procedures for producing some degree of narcosis and insensibility to pain by surgeons in much earlier times, Hugo stressed the use of inhalations of varied and secret drugs as anesthetics. The composition of his "spongia somniferens" was known only to him. By many it is presumed that this method of narcosis was originated by him. Whether this is true or not we do not know, but at any rate he popularized its use.

His therapeutic armamentarium is evidence of his versatility. He himself made many "collyria," prescribing them for redness and inflammation of the eyes. A "holy salve" which was a "cure all" in the healing of wounds, was a secret formula. Another salve contained mercury which he used in skin diseases and it is to him that credit must be given for the first description of the salivation and gum changes produced by the prolonged use of mercury. Others of his salves contained arsenic and were used as cauterizing agents in fissures and wounds. These were called "encaustrum" and likewise were a secret composition of his own.

He wrote of using a "coagulum leporis" which historians relate was not new to him or his time, having been used in earlier days. It consisted merely of the coagulated blood of hares. If only these historians had recorded in what conditions he used this coagulated rabbits' blood, particularly if in hemorrhage, what an interesting point that would make. He did describe extensive bleeding from wounds, but no technique of treatment other than his usual method of closure.

An anodyne powder is described by his son as being a part of the after treatment of all his severe cases, but its formula is not mentioned. It was called only a "wound drink," dissolved in wine.

Like any good surgeon he stressed the after care of his patients and paid particular attention to their diet. "Pigmenta," his wound drinks were called because they were designed to produce blood and flesh. He was a very pious individual and instructed his patients to say a prayer whenever they partook of these drinks, which was several times a day. He even designated certain prayers which should be said at the various hours. These wound drinks were mainly highly concentrated beef or chicken broths.

As to meats, he declared that capons, partridges, pheasants and small birds with fine beaks, were of exceptional quality to produce blood and sinew. Meat of suckling

goats and of castrated spring cattle was also very fine. Chicken eggs were to be eaten daily, cooked in any manner except sodden or fried.

Paramount, however, was the instruction that the best wine obtainable, not requiring the addition of water, should be taken for regaining strength.

That he took part in the civic life about him is evidenced by the fact that in 1214 he was appointed city physician to the town of Bologna. In 1218 he was persuaded to join the Bolognese contingent to the Crusade, returning in 1221 to take up once again his duties as city physician. In this post he is credited with drawing up the first legal sanitary measures and rules regarding water, waste disposal, etc., as a modern health officer would be expected to do.

It is rather singular that a great name and presumably a great surgeon should have singled out the name of Hugo of Lucca upon which to vent his vilification and abuse. Guy de Chavliac swore to the high heavens that not only Hugo but also his son Theodorich, were no more than charlatans and quacks of the highest order, and accused them of plagiarizing the older Greek masters, then claiming for themselves that all they propounded was original.

This anger and abuse was probably incited by the fact that Guy de Chaviliac was the great exponent in his time of the theory of "laudable pus," while Hugo and his son proclaimed vehemently that there was no such thing. "It is not necessary, as many supposedly great minds have taught and are teaching, that pus must be generated in wounds. To practice such a method is only to hinder nature and to prevent good approximation and healing of wounds."

Yet we find in the works of Guy de Chaviliac, who accuses them of plagiarism, the identical descriptions word for word as written by Theodorich at the direction of Hugo, and even the illustrations to the chapters of his great opus, his surgery, are copies of those from the works of Hugo.

A perusal of the life and work of the two individuals makes one ponder whether one shouted the loudest and had the best publicity while the other went on unspectacularly producing results. At any rate, history seems to have given the verdict of popularity to Guy de Chavliac, whether he deserved it or not.

The School of Bologna itself, independent of its famed faculty member, was instrumental in contributing a milestone in the progress of medical science and it came about in an interesting manner. First, in speaking of these medieval universities certain things must be kept in mind in order that a better picture may be obtained. In Bologna as at Salerno, Monte Cassino, and other centers, the actual popularity and success of the institution was dependent entirely upon the fame and learning of its faculty. It was because of the quality of scolarship that students flocked from England, Germany, France, and other countries to any one particular center. At the very peak or height of its affluence the school of Bologna had a registration of 10,000, speaking volumes for its faculty. Its fame rested upon its school of medicine and surgery and its school of law.

One day in the streets of Bologna a man dropped dead from no apparent cause. The medical faculty being unable to give any satisfactory explanation other than that it was probably due to some disease of the heart, the law faculty insisted that a postmortem examination of the heart should be made and this was done.

It is rather certain that human dissection was practiced in old Grecian and Roman times, but it was done secretly and no actual record ever was made. There is an old medical manuscript in the Bodleian Library which contains a drawing of a dissection scene, using a human body. This manuscript is presumed to have been written about the year 1300 A.D. The Church had formally prohibited human dissection because it had become the practice of the Crusaders to cut up the bodies of those who died and boil the pieces in order

to separate the flesh from the bones, since it was more feasible to repatriate the bones of those who died in distant countries. The bones might then be buried in the home parish. Transportation facilities had to be considered. It was easier to transport a few bones than a whole body, but the Church forbade this practice.

When the law faculty considered this case of sudden and unexplained death, they demanded at least a post-mortem examination of the heart. By so doing they broke the fetters of custom which had entwined medical anatomy for ages and unknowingly laid the foundation for the school of brilliant anatomists who were to follow and who were to base their descriptions and drawings upon the study of actual human bones and flesh. This was definitely a milestone in medical progress, and to the University of Bologna must be given the credit first for the establishment of post-mortems in the study of disease, and second, for the brilliant strides in anatomy which took place from that time on.

As to the writings and works which Hugo left to posterity, as stated previously, there is some degree of uncertainty as to whether much has disappeared or whether he really wrote little. He lived to a very old age, reckoned by biographers to have been in the vicinity of one hundred years. He had four sons, all of whom were physicians, but only one, Theodorich, attained any degree of fame. To him Hugo entrusted all of his secret formulae and methods of therapy, and it is recorded that he did not impose the oath of secrecy upon this son as was the custom of the times. This custom originated with earlier physicians and surgeons in order to protect the formulae and methods they used for the benefit of their families when they died, that they might continue to derive remuneration from them. If there was a son who elected to become a physician all information was given to him and he was sworn to secrecy. If there was no son and an assistant had proved of sufficient loyalty and merit, then the information was his on the same basis.

It is probable that Hugo wrote little, leaving this part of his work to his son. A "Cyrurgia" was published in Venice in 1497, and is presumed to consist of the combined manuscripts of Theodorich and Hugo. It is this work which Guy de Chavliac later incorporated into his own. Later editions of it were published in Venice, in 1499 and 1500. Two treatises which were definitely known to exist in manuscript from, a "De Sublimatione Arsenici," and a "De Aluminibus et Salibus" are presumably lost.

Copies in Spanish and Italian exist of a work entitled "Practica Equorum" dealing with veterinary medicine. It is supposed that the material in this is entirely Theodorich's.

Only the "Cyrurgia" remains to Hugo's memory. He compiled it before the middle of the thirteenth century and dedicated it to the bishop of Valencia. This fact may account for its later Spanish translation. From this Spanish translation, one was made into Hebrew.

A small manuscript dealing with the manufacture of wines and with their use in disease was known to have existed but its whereabouts today is unknown. If found, it would probably be the first treatise on wine, antedating Andre De Villanouas' "De Vinis" by some years.

Hugo should be classed among that large group of physicians and surgeons who have stood unflinchingly by principles in which they believed. His place in medical history is secure because of his intensely practical aptitude, his ability as a surgeon, as a teacher of medicine, and as the real founder of what could be called the Italian school of surgery which had originated with him at Bologna in the middle of the thirteenth century.

Surgery of the Gall-
Bladder and Extrahepatic
Bile Ducts

BY

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INTRODUCTION

SURGERY of the gall-bladder and extrahepatic bile ducts represents one of the most common fields for surgical practice. There can be little doubt that the need for surgical procedures in this field is increasing because of the extension of life, whereby more persons reach the age when cholelithiasis is both likely to occur and prone to produce its characteristic symptomatology. In 1850 persons over 65 years of age comprised 2.6 per cent of our population; today they constitute 6.4 per cent; and in 1980, according to present progression, they will have reached 14.4 per cent. The surgery of the biliary tract, therefore, will become an even more important consideration for the coming generation of surgeons.[19]

This discussion is planned to give a practical approach to the surgical treatment of the common lesions of the gall-bladder and extrahepatic bile ducts and, therefore, our primary concern must be the removal of an acutely or chronically inflamed gall-bladder usually associated with calculi. But since approximately one-fifth of the patients requiring cholecystectomy have calculi in the extrahepatic biliary ducts, procedures involving the common duct become of equal importance. Any surgeon who dares subject his patient to cholecystectomy must be qualified to recognize and carry out safely the procedures best designed to remove or correct pathologic changes involving the extrahepatic ducts as well. In addition, a small miscellaneous group of conditions of less frequent occurrence will be presented, including congenital anomalies, division of the duct or traumatic stricture, and compression of the ducts by intrinsic or extrinsic tumors. This treatise will not cover primary disorders of the liver unless extension of the primary condition involves the extrahepatic biliary system. Thus, abscess, tumors, and cysts of the liver will not be referred to.

It is not out of place in a treatise of this kind to call to attention the congenital lesions since some may persist to later life and thus unhappily surprise the unwary surgeon. The common congenital anomalies are of the gall-bladder and of the common duct.

A double gall-bladder has been reported twenty-eight times. This accessory bladder may be contiguous to the normal organ, have an opening directly into the gall-bladder or into the normally formed gall-bladder's cystic duct, or the accessory gall-bladder may be at some distance from the normally situated viscus and empty into a totally different radicle of the hepatic duct.

Bilobed gall-bladders occur and gall-bladders with pouches much like diverticula. Such accessory gall-bladders may cause no symptoms until they become the seat of either inflammation or stone. Should such an accessory gall-bladder be on a freely movable pedicle, torsion may occur and an abrupt clinical syndrome be brought to the surface. The clinical picture of such torsion is characterized by abrupt and continuous pain of great severity set aside from biliary colic by its steady persistence and failure to show periodicity and remittance. Nausea and vomiting usually occur.

Anomaly of the common bile duct may take on several forms, the commonest clinical syndrome being that known as cystic or idiopathic dilatation of the common bile duct. This may be due to failure of the duct to develop, stenosis at the papilla, congenital weakness of the duct wall, the abnormal course of the duct, or even valve-like obstruction, such as occurs on rare occasions in the prostatic urethra. Apparently cystic dilation of the bile duct is a well established clinical syndrome appreciated by those surgeons familiar with the surgery of childhood. The prominent findings are tumor, jaundice, and pain. Experience has shown that it is unwise to search for the cause of this idiopathic dilation of the common duct and wise to perform at once an anastomosis usually between the dilated duct and the duodenum. In a report by Gross of fifty-two cases, the mortality of surgical treatment for the whole group was 69 per cent, but in those treated by anastomosis between the biliary tract and intestine the mortality was only 9 per cent.

Other anomalies of the common duct occur. Boyden reports sixteen cases of congenital doubling and five cases of abnormal insertion of the common duct. Ladd has called attention to complete

atresia of the duct below the insertion of the cystic duct and also to stenoses occurring in the hepatic ductile system. As a matter of interest the common duct may be entirely absent and its failure of development may extend well up into the hepatic radicles. What is important in this consideration is that the surgeon recognize the fact that anomalies occur in order that he may be forewarned in atypical cases.

CHRONIC CHOLECYSTITIS WITH AND WITHOUT STONE

An analysis of the symptoms of the patients in the Peter Bent Brigham Hospital subjected to surgery for chronic cholecystitis with or without cholelithiasis has been made in an effort to throw some light on the symptoms of those subjected to cholecystectomy. Our records show that approximately 90 per cent have a history of biliary colic and pain of marked severity. This indicates that, in general, we have declined to operate upon patients who have only vague indigestion as the major complaint because it has been generally demonstrated that the results do not justify the risk.[7,11] Two-thirds of our patients with chronic cholecystitis and cholelithiasis have pain in the right upper quadrant referred to the back. Not more than one-third have at any time complained of epigastric distress; in contrast, patients with acute cholecystitis or common duct stone show a much higher instance of epigastric distress. Approximately 10 per cent of our patients have pain in the left upper quadrant or left infrascapular region instead of the customary right upper quadrant, due, some believe, to an associated pancreatitis. Rehfuss and Nelson report a similar figure. We are unable from experimental observations to explain to our satisfaction the mechanism of left upper quadrant pain.[27]

If vomiting is spontaneous and pronounced when associated with gallstones, we believe that a calculus is located within the cystic or common ducts. This supposition is borne out by an analysis of our cases. Vomiting was found to occur in less than half the cases of chronic cholecystitis in which the patients gave no history of jaundice and in which no stone was found in the ampulla or the cystic or common ducts. This finding agrees with the views of those who believe that a patient with cholelithiasis uncomplicated by duct obstruction may have nausea and induced vomiting but not involuntary vomiting.

A high percentage (94 per cent) of the patients subjected to cholecystectomy at the Peter Bent Brigham Hospital have a proved calculus at the time of operation. This high incidence of calculus no doubt results in part from our rigid selection of patients, who must have pronounced gall-bladder symptoms, such as colic, etc., as previously described, and in part from our custom of submitting every patient to intravenous cholecystography before cholecystectomy unless calculi are positively shown by the oral method. A poorly functioning gall-bladder or failure of the gall-bladder to be visualized by the oral test is not sufficient evidence, in our opinion, to warrant cholecystectomy unless the clinical symptoms are overwhelmingly in favor of disease of the biliary system. It has been shown by Sosman that approximately 30 per cent of the gall-bladders which appear to be abnormal with oral administration of dye, single bottle technique, can be visualized by giving the dye intravenously.

The following technique is followed for the intravenous administration of tetraiodophenolphthalein. A standard dose of 2.5 gm. of tetraiodophenolphthalein is given to all patients weighing over 50 Kg. (110 pounds); patients weighing less receive 40 mg. of the dye per Kg. of body weight. The dye is diluted in 30 c.c. of distilled water; autoclaved for fifteen minutes; diluted with 100 c.c. of sterile saline; and given intravenously about 11 P.M. No food is allowed until after the x-ray, but water is permitted as desired. A soapsuds enema is given. Roentgenograms are taken in the morning some ten hours after administration of the dye.

It is interesting to note that only about one-third of the patients seen in the Roentgenological Department showing pathology in the gall-bladder by the dye test are subsequently submitted to surgery. This does not mean necessarily that surgery is not recommended, but that certainly a large percentage of these patients not coming to operation must get along comfortably without surgical intervention.

With such rigid selection of cases for surgery, the failures from improper interpretation of symptoms from the biliary tree or from confusion with an associated arthritis of the spine or pathology of the right kidney, duodenal ulcer, or other factors are infrequent; moreover, operation is limited to the group of patients that statistics show will receive the greatest benefit from surgery.

Since the patient with chronic cholecystitis can usually have the operation at a time of election, he should receive adequate

preoperative preparation. Obese persons with symptoms severe enough to warrant cholecystectomy should be held rigidly to a low caloric diet until the weight is substantially reduced, because mortality in the obese is distressingly high. Otherwise a high carbohydrate intake is provided, and our patients are urged to eat candy in any form, cereals, and similar foods for several days previous to operation. In addition, it is desirable to force fluids for the twenty-four hours before operation. The high incidence of pulmonary complications following any upper abdominal surgery makes it imperative that the patient be free of chronic cough and respiratory infection so far as this is possible. As a check on the respiratory condition our patients have vital capacity determined. This, if abnormally low, calls for cancellation of the surgical procedure until the cause and possible correction of this low vital capacity can be determined. Phlebitis or other infection about the lower extremities must be cleared up before operation because of the all too frequent occurrence of embolism in association with gall-bladder operations. For this reason the veins of the lower leg are not used if difficulty is experienced in finding the veins of the cubital fossa when administering solutions or dye. Since many patients have difficulty in voiding after any type of operation, each patient during the preoperative days should familiarize himself with the use of the bedpan, especially for urination.

The condition of the patient and the custom of the surgeon govern the choice of anesthesia, but adequate relaxation of muscles simplifies the operation. In elderly or debilitated patients, local anesthesia (novocaine, 1 per cent, without adrenalin) may be used with satisfaction. However, the majority of our patients receive avertin supplemented by ether; if the patient is jaundiced or if there is evidence of liver damage, avertin is not given.

Cholecystectomy, Retrograde Method. The importance of placing the patient on the operating table in the position most advantageous for later exposure and for obtaining maximum benefit from natural and artificial light cannot be overemphasized. The patient is hyperextended at the costal margin either by elevating the gall-bladder rest (Fig. 1) or by breaking the operating table. (Fig. 2.) The entire table is tilted until the patient is in a semi-erect position. With the patient in position the liver and gall-bladder tend to drop below the costal margin and at the same time the intestines fall away from the upper abdomen, giving natural retraction for the

field of operation. With side-lighting in the operating room the operating table is turned so that the feet of the patient point toward the maximum point of natural light. This is augmented by overhead
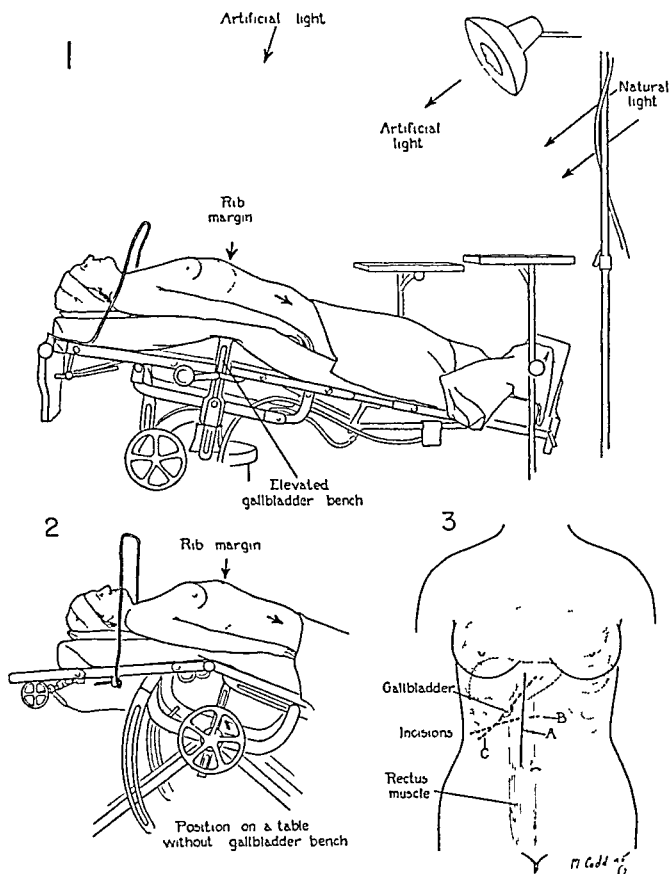
FIGS. 1 AND 2. The correct position of the patient on the operating table is shown.

FIG. 3. The common types of incisions for cholecystectomy are indicated.

artificial light, preferably supplemented by a portable spotlight which can be shifted to meet the demands and problems of the operation as it progresses. (Fig. 1.) Two instrument tables of the Mayo type are brought close to the operating field over the lower extremities of the patient.

After the surgeon has assured himself that the position and lighting are entirely satisfactory, the skin is prepared with the antiseptic of choice. We prefer to use alternately alcohol (70 per cent

ethyl) and zephiran (1:1000 aqueous solution).[9] The site of incision is then scratched before the field is entirely covered with sterile towels and drapes. Although various types of incision (A, B, and C)
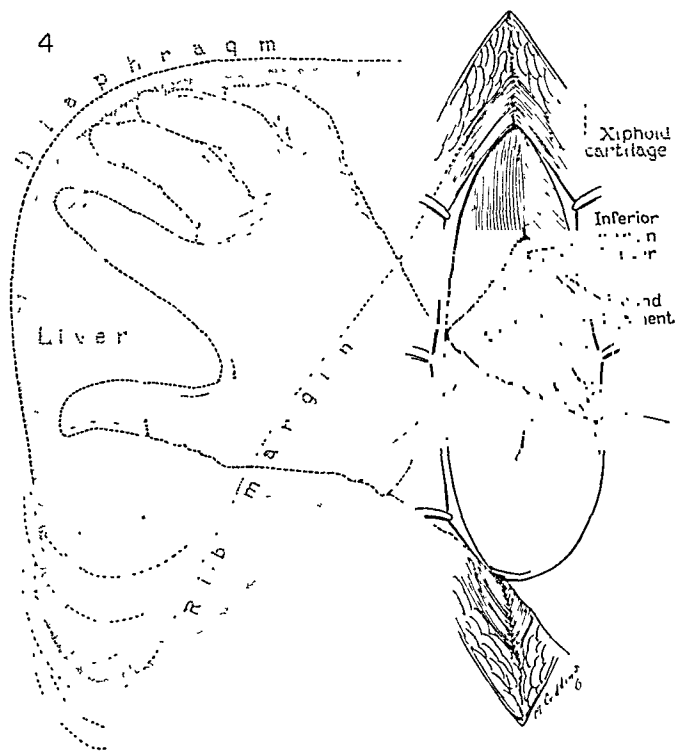
FIG. 4. The right hand of the surgeon is passed over the liver to aid in displacing it downward.

are shown, we usually employ a vertical, high right rectus incision (A), as shown in Figure 3. This is outlined starting over the costal margin just to the right of the ensiform process and continuing downward to the umbilicus or below depending upon the size of the patient. A liberal skin incision is always made to make certain that it is longer than the peritoneal incision. After the incision has been outlined with the back of a scalpel, the patient is draped with towels so that all skin is covered except the outline of the incision itself. The final drapes are applied.

When the incision has been deepened to the rectus sheath and all bleeding points have been tied, the fat is protected from contamination by towels or pads moistened with warm, isotonic salt solution. The rectus sheath is incised in its midportion or toward the midline, and the muscle fibers are split in a parallel fashion.

Some surgeons prefer to reflect the entire rectus muscle laterally to avoid injury to the motor nerves. Active bleeders are ligated at each tendinous striation. If the rectus muscle has been divided in its midportion, an effort should be made to retract the nerves downward or upward to avoid their permanent damage. The transversalis fascia and peritoneum are then opened, beginning to the right of the xiphoid and continuing to the umbilicus. At this point the underlying falciform and round ligaments are encountered and retracted medially, following which the transversalis fascia and peritoneum are fixed with curved half-length clamps to the gauze packs which already cover the subcutaneous fatty tissue. The incision is carried upward to the region of the xiphoid, for in our experience the pleural cavity has never been opened when this approach for cholecystectomy was used. (Fig. 4.) An incision properly made exposes the anterior surface of the liver, making possible an easier presentation of liver and gall-bladder. If the incision is started at the costal margin over the region of the gall-bladder instead of near the midline adjacent to the xiphoid, the procedure is much more difficult.

After the peritoneal cavity has been opened, the gloved hand moistened with warm salt solution explores the abdominal cavity unless there is acute suppurative infection involving the gall-bladder. The stomach and pylorus especially are inspected and palpated. Following the general abdominal exploration, the surgeon passes his right hand up over the dome of the liver to allow displacement of air between the diaphragm and liver and to aid in dislodging the liver downward. (Fig. 4.) When assistance is limited, a self-retaining retractor of the Balfour type may be used advantageously or an ordinary retractor of the Halsted type may be used on the right side to retract the costal margin. A half-length clamp is applied to the round ligament and another to the fundus of the gall-bladder. (Fig. 5.) Some surgeons prefer to divide the round ligament between half-length clamps; usually, however, this is unnecessary. Traction is then maintained downward by the clamps on the fundus of the gall-bladder and on the round ligament. This traction is exaggerated with each inspiration as the liver is projected downward. (Fig. 5.) After the liver has been pulled downward to the limit of easy traction, the half-length clamps are then pulled toward the costal margin to present the under surface of the liver and gall-bladder. (Fig. 6.) These clamps are then held by an assistant, while the surgeon prepares to wall off the field. Should the
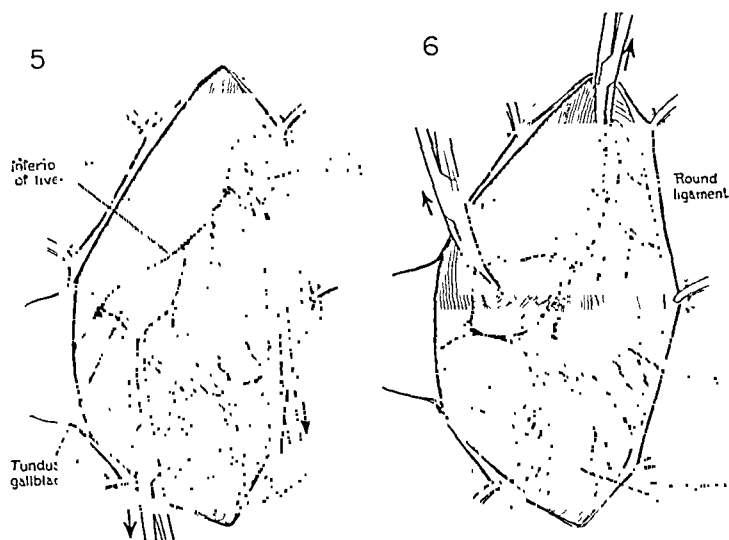
FIG. 5. Traction is applied to the round ligament and fundus of the gall-bladder with half-length clamps to pull the liver downward.

FIG. 6. Traction on the half-length clamps applied to the round ligament and gall-bladder is directed outward and upward to expose the under surface of the liver and gall-bladder.
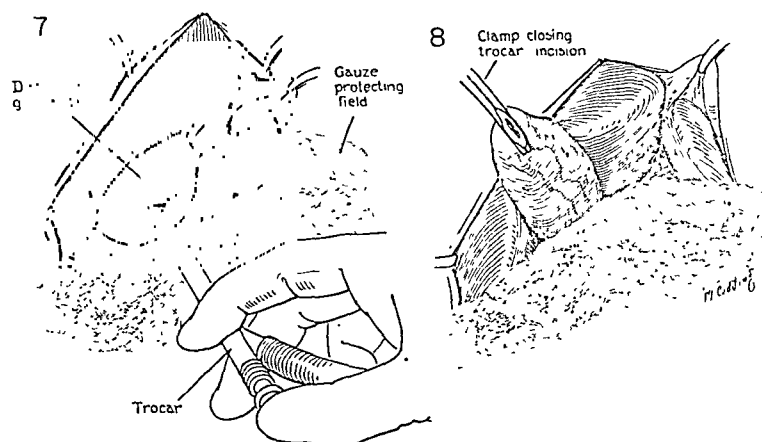
FIG. 7. The contents of an acutely distended gall-bladder are aspirated with a trocar before the application of clamps to the gall-bladder.

FIG. 8. A curved half-length clamp is applied to the fundus of the collapsed gall-bladder to seal the trocar opening. The concavity of the clamp is toward the costal margin.

gall-bladder be acutely inflamed and distended, it may be necessary to aspirate some of its contents through a trocar before the half-length clamp is applied to the fundus, lest small stones be forced into the cystic and common ducts. (Figs. 7 and 8.)
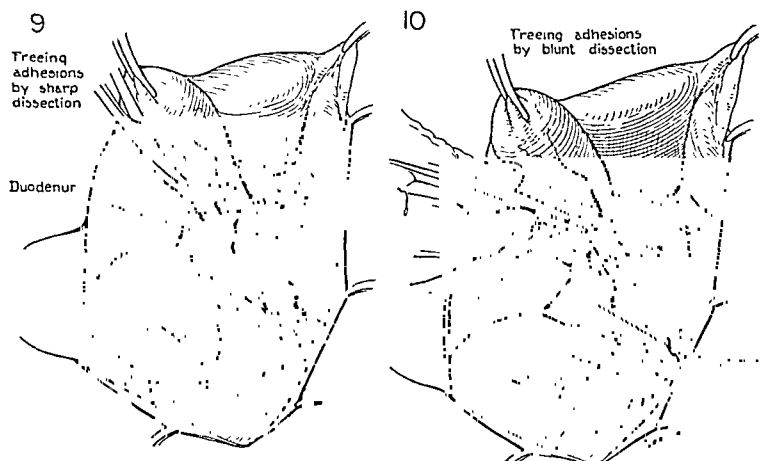
FIG. 9. Any adhesions between the under surface of the gall-bladder and adjacent structures are divided by sharp dissection.
FIG. 10. Following sharp dissection the region of the ampulla may be further exposed by gauze dissection.

Adhesions between the under surface of the gall-bladder and omentum are frequently found drawing the duodenum or transverse colon up into the region of the ampulla. These adhesions are divided with curved scissors until an avascular cleavage plane can be developed adjacent to the wall of the gall-bladder. (Fig. 9.) After the initial incision is made, it is usually possible to brush these adhesions away with gauze sponges held in forceps, thereby presenting a smooth gall-bladder wall. (Fig. 10.) Once the gall-bladder is freed from its adhesions, it can be lifted upward, thus affording better exposure. In order that the neighboring structures may be packed away with moist gauze pads, the left hand of the surgeon is inserted into the wound palm down to direct the gauze pads downward as they are introduced with long, smooth forceps. (Fig. 11.) The stomach and transverse colon are packed away, and a final gauze pack is inserted into the region of the foramen of Winslow. (Fig. 12.) The gauze pads are held in position either by a wide, smooth retractor along the lower end of the field (Fig. 13) or preferably by the left hand of the first assistant, who with fingers slightly flexed and spread apart maintains moderate downward and slightly upward

11

12

Introduction of
gauze to wall off
operative field

Gauze covering
hepatic flexure
of colon
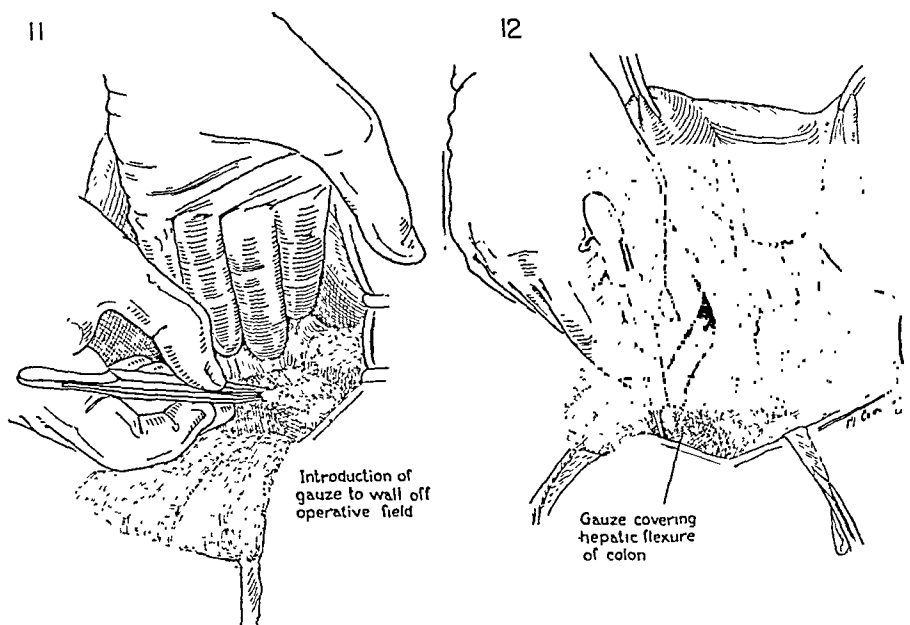
FIG. 11. Two gauze pads are necessary to pack off the stomach and transverse colon.
FIG. 12. A gauze pack is placed to wall off the right lumbar gutter.

13

Round
ligament

Hepato-
duodenal
ligament

Gauze in
foramen of
Winslow

Balfour
self-retaining
retractor

14

Round ligament

Medium
abdominal
retractor

Alternate
method of
retraction

Left hand of
first assistant
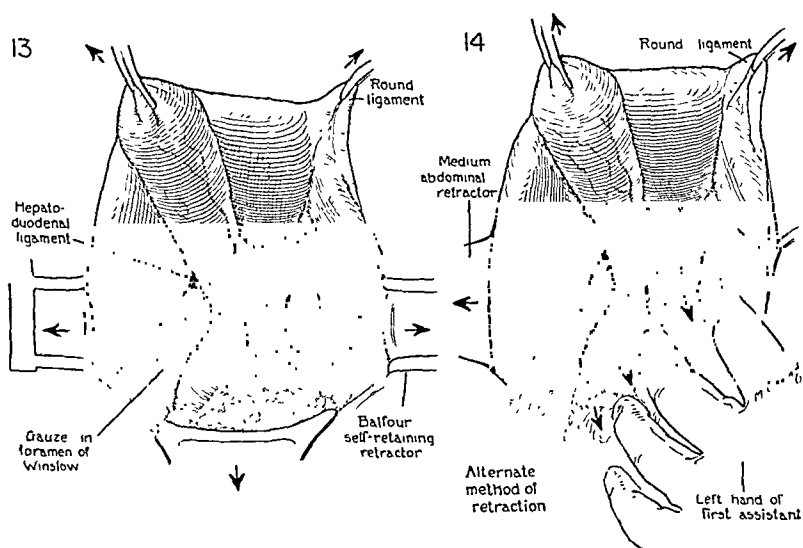
FIG. 13. Exposure is maintained by a self-retaining Balfour retractor supple-
mented by a Halsted retractor at the lower angle of the wound.
FIG. 14. The alternate method of retraction is obtained by a lateral abdominal
retractor and the left hand of the first assistant.
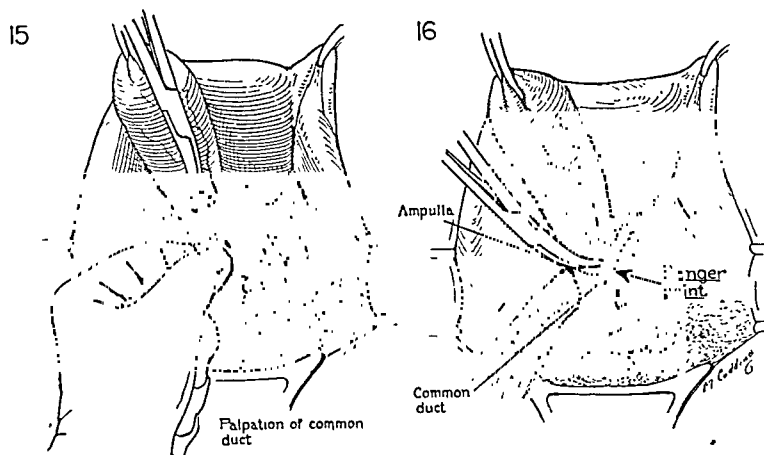
FIG. 15. The cystic and common ducts are palpated for calculi. Additional
traction is applied to the gall-bladder by a curved half-length clamp
placed above the ampulla.

FIG. 16. A curved half-length clamp must never be applied low in the
region of the ampulla of the gall-bladder because of the danger of includ-
ing part or all of the common duct.
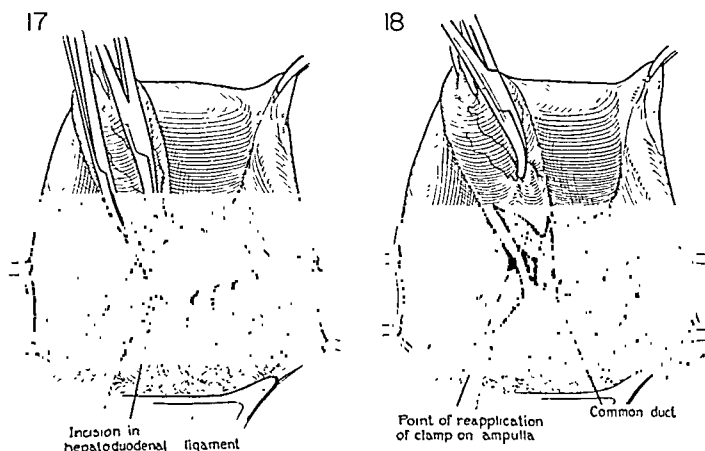
FIG. 17. The region of the ampulla and cystic duct is exposed by an
incision in the lateral margin of the hepatoduodenal ligament. This
may be made with scalpel or long curved scissors.

FIG. 18. The incision in the hepatoduodenal ligament is carried to the
upper side of the ampulla and exposes the common duct. A curved
half-length clamp may now be safely applied to the ampulla at the
location marked x.

pressure, better defining the region of the gastrohepatic ligament. (Fig. 14.)

After the field has been adequately walled off, the surgeon introduces his index finger into the foramen of Winslow and with
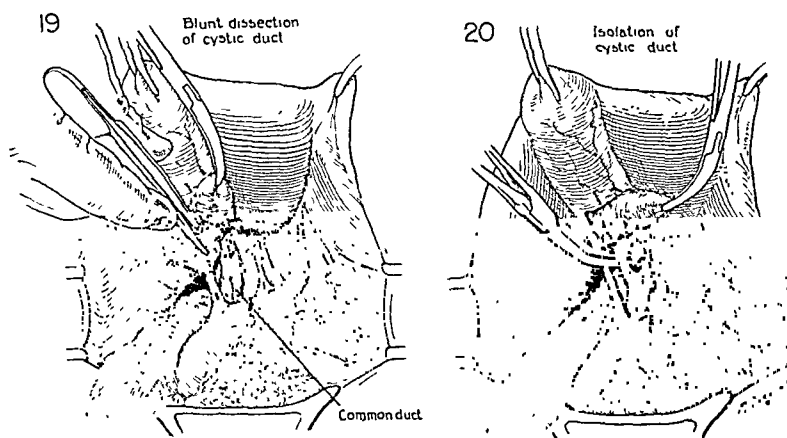
FIG. 19. Traction is maintained on the ampulla of the gall-bladder while the cystic duct is dissected by blunt dissection.
FIG. 20. A long Lower clamp is used to isolate the cystic duct from adjacent structures.

finger and thumb thoroughly palpates the region of the common duct for evidence of calculi as well as for thickening of the head of the pancreas. (Fig. 15.) A half-length clamp grasps the under surface of the gall-bladder to maintain upward traction. The early placement of clamps in the region of the ampulla is one of the frequent causes of accidental injury to the common duct. This is especially true when the gall-bladder is acutely distended, because the ampulla may run parallel to the common duct for a considerable distance. If the clamp is applied low and blindly, part or all of the common duct may be included in it. (Fig. 16.) For this reason it is always advisable to divide the peritoneal attachment in the region of the ampulla rather high on the gall-bladder, extend the incision carefully downward to the hepatoduodenal ligament, and by means of blunt dissection free up the region of the ampulla. (Fig. 17.) The peritoneum is usually incised with long Metzenbaum dissecting scissors. An incision is likewise made in the peritoneum on the upper side of the ampulla and cystic duct. (Fig. 18.) By blunt dissection the gall-bladder wall is followed down to the region of the cystic duct. Dissection is gingerly carried out in this region because of possible damage to the cystic artery. After the peritoneum has been incised

and the ampulla of the gall-bladder has been more clearly defined, the half-length clamp may be reapplied lower on the ampulla in the location, x. (Fig. 18.)
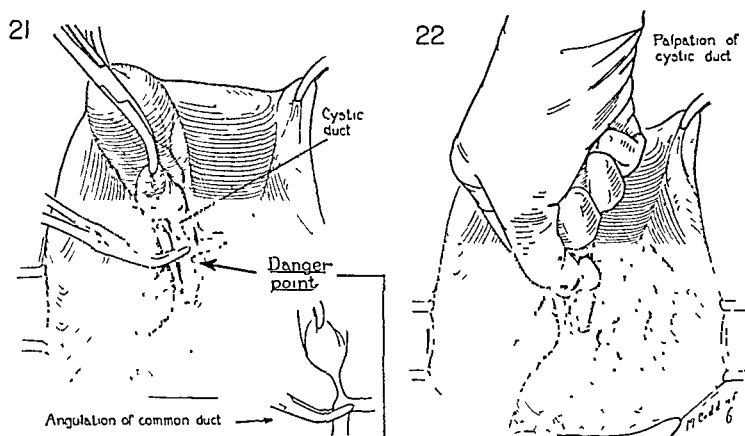
FIG. 21. The cystic duct must not be clamped until its relation to the common duct is visualized. Excessive traction on the ampulla angulating the common duct may be responsible for accidental clamping of the latter structure.

FIG. 22. The cystic duct is palpated carefully for calculi before clamps are applied.

With traction maintained on the ampulla, the cystic duct is defined by means of gauze dissection. (Fig. 19.) A long Lower clamp is then passed behind the cystic duct and moved to and fro, its blades cautiously separated, until the cystic duct is well isolated from the common duct. (Fig. 20.) Whenever possible, unless occluded by a severe degree of inflammation, the cystic duct and cystic artery should be isolated separately to permit individual ligation. Some surgeons prefer to isolate and ligate the cystic artery before ligating the cystic duct; however, it is our custom to isolate and divide the cystic duct first. Under no circumstances should the surgeon blindly apply a right-angle clamp to the supposed region of the cystic duct in the hope that he can include both in one mass ligature. If the upward retraction on the gall-bladder is marked and the common duct is quite flexible, it is not uncommon to have it angulate acutely upward giving the appearance of a prolonged cystic duct. The surgeon must recognize this possibility and con-stantly keep this mental image before him to avoid injury to or division of the common duct when the right-angle clamp is applied to the supposed cystic duct. (Fig. 21.) It is advisable, after the

cystic duct has been isolated, for the surgeon to palpate it thoroughly to make certain that no calculi are forced into the common duct by the application of clamps and that none is left in the stump
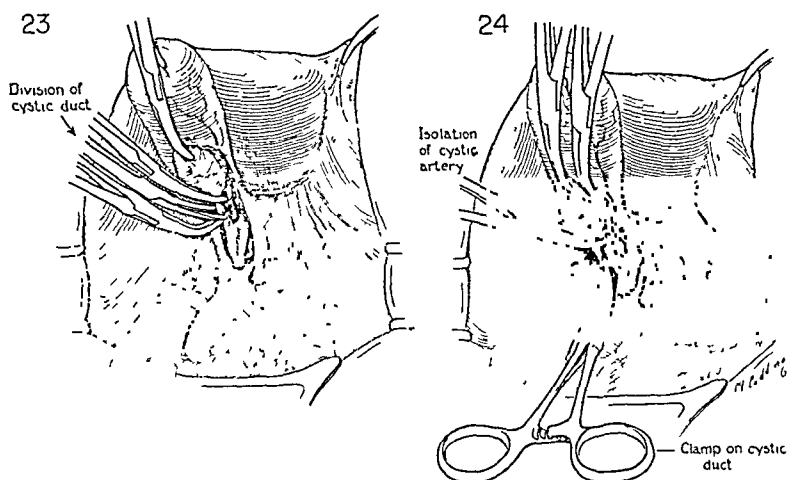
FIG. 23. A curved half-length clamp is used instead of a second right-angle clamp to give sufficient room for division of the cystic duct.
FIG. 24. The long Lower clamp is alternately opened and closed to isolate the cystic artery.

of the cystic duct. (Fig. 22.) Palpation rather than the application of a clamp in an upward and downward motion is the more judicious maneuver because of the danger of crushing small calculi in the cystic duct. Their resultant extrusion into the common duct might offer a possible nucleus for the later complication of common duct stone.

The size of the cystic duct must be carefully noted by the surgeon. If the cystic duct is dilated and it would seem from palpation that the calculi in the gall-bladder could pass easily through it, the surgeon is in duty bound to perform a choledochostomy. Regardless of whether or not the patient is jaundiced or whether he believes from the history that the patient has apparently no indication of a common duct stone, the size of the common duct and the consistency of its wall should be determined in every case in which the gall-bladder is removed. If after palpation of the cystic duct the operator is certain that the common duct is not so angulated as to be included in the clamp and that it need not be explored, a long Lower clamp is applied. (Fig. 23.) Because it is more difficult to divide the duct between two right-angle clamps closely applied, we prefer to place a half-length clamp above the initial right-angle

clamp. The general curvature of the half-length clamp makes it ideally suited for directing the scissors downward at the subsequent division of the cystic duct. Should it seem judicious to explore the
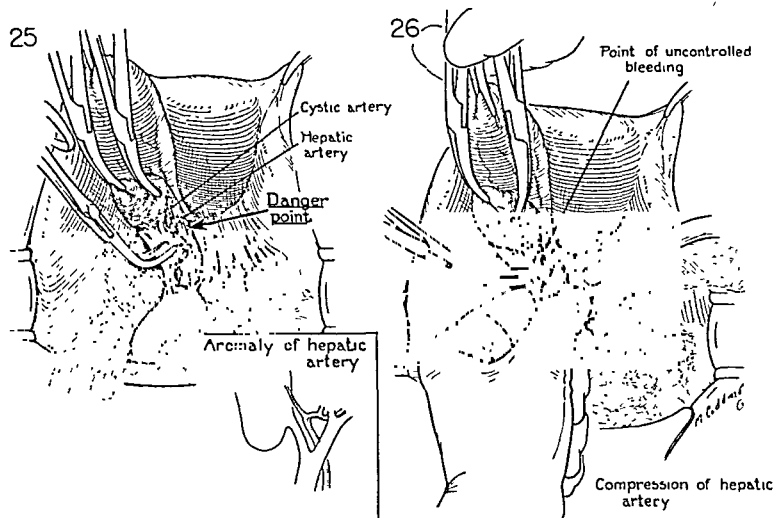
FIG. 25. The cystic artery is not in a constant location. The right hepatic artery may be accidentally ligated with subsequent death of the patient.

FIG. 26. The left hand of the surgeon may be used as a tourniquet to the hepatic artery in cases of uncontrolled bleeding from the cystic artery.

common duct, a right-angle clamp is not applied to the cystic duct nor is it divided until after the common duct has been explored.

If the cystic artery was not divided before the cystic duct, it is now isolated with some surrounding tissue by a clamp similar to that used in isolating the cystic duct. (Fig. 24.) Except in cases of acute cholecystitis, the cystic artery is usually a small vessel. The surgeon should keep in mind the possibility of accessory vessels in the region of the cystic duct and should be familiar with the anomalous positions and routes sometimes taken by the right hepatic artery (Insert, Fig. 25). A clamp should never be blindly applied to this region because occasionally the hepatic artery is in an anomalous location and unwittingly may be clamped and divided with the resultant death of the patient. We have never felt confident in attempting to determine the location of the cystic artery by palpation for pulsation. If the clamp or tie on the cystic artery slips off, resulting in uncontrollable bleeding, the hepatic artery may be compressed by the thumb and index finger of the left hand, temporarily controlling the bleeding; the field may be dried by the assistant; and then as the surgeon releases his compression of the

hepatic artery, a hemostat may be safely applied to the initial bleeding point. (Fig. 26.) If a satisfactory exposure has been obtained and the dissection has been carefully carried out, the region
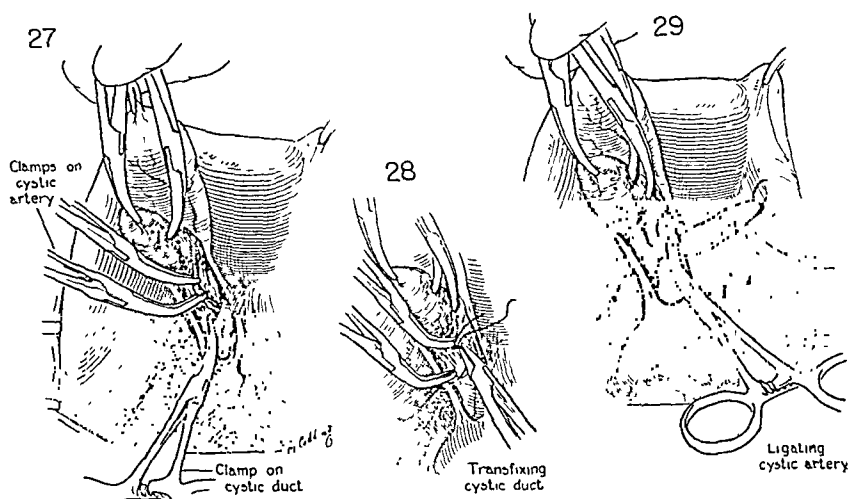
FIG. 27. The cystic artery is divided between a Lower clamp and curved half-length clamp.

FIG. 28. The stump of the cystic duct is ligated with a transfixing suture of fine silk.

FIG. 29. A fine silk ligature is applied to the cystic artery. Note that the cystic artery and cystic duct are not tied en masse.

of the cystic artery can be easily defined. Here again first a Lower clamp and then a half-length clamp is applied, and the artery is divided between them (Fig. 27) and tied with a fine silk ligature. (Fig. 29.) The cystic duct is then tied by passing a French needle through the very edge of the duct, not through its lumen, to permit a transfixing suture of fine silk to be applied. (Fig. 28.) We do not feel that it is necessary to cauterize the stump of the cystic duct after it has been divided. The stumps of cystic artery and cystic duct are each thoroughly inspected, and the common duct is again visualized to make certain that it is not angulated or otherwise interfered with.

After the cystic duct and artery have been tied, removal of the gall-bladder is carried out. About 1 cm. from the liver edge an incision is made with a scalpel into the serosa but not into the lumen of the gall-bladder itself. (Fig. 30.) It is important that the serosa be divided with a scalpel or scissors so that the gall-bladder is not peeled from its bed by traction, thereby leaving raw liver surface, a frequent happening when exposure is difficult in the case of a small, shrunken

gall-bladder. Moreover, it is always advisable, and usually possible, to leave a liver bed which can be peritonealized, thus overcoming the possibility of subsequent bile leakage and the necessity of
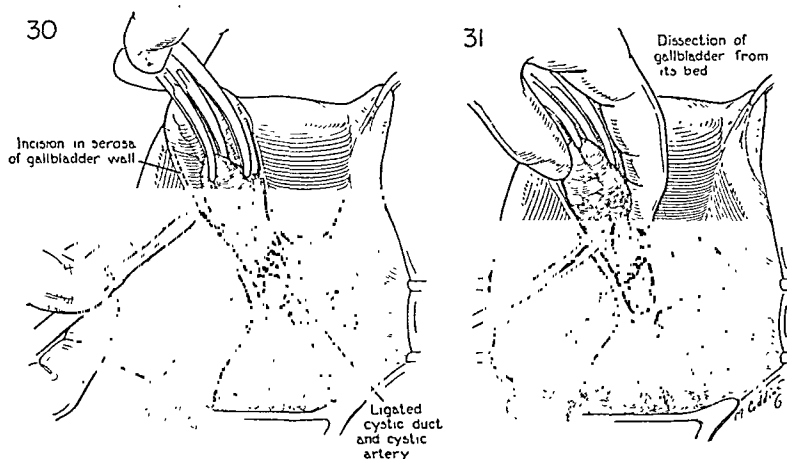
FIG. 30. An incision is made in the serosa of the gall-bladder in preparation for its subsequent enucleation.

FIG. 31. The gall-bladder is grasped in the left hand of the surgeon while it is dissected from its bed by sharp dissection.

inserting a cigarette drain. At this stage the gall-bladder is controlled by the left hand of the surgeon. The clamps applied to the region of the cystic duct and fundus are grasped in the left hand of the surgeon and retracted outward while a cleavage plane is maintained and fine adhesions are divided on the liver side of the gall-bladder. (Fig. 31.) After a cleavage plane has once been developed by sharp dissection, it is frequently possible to enucleate the gall-bladder from its bed in an avascular cleavage plane by inserting the index finger upward between it and the liver bed. (Fig. 32.) The gall-bladder should never be completely removed from its bed, however, but should be retained to serve as a retractor until the peritoneal margins in the deep portions of the wound are approximated. (Fig. 33.) The gall-bladder is finally removed, when the last stitch is ready to be inserted at the liver edge. (Fig. 34.) A warm, moist gauze sponge is then laid along the suture line to make certain that there is no oozing and that the field is absolutely dry. It is advisable at this point to lower the gall-bladder bench, if it was used, before proceeding with the closure. If the appendix can be delivered easily into the field, it is removed after the desired procedures on the biliary tract have been completed.
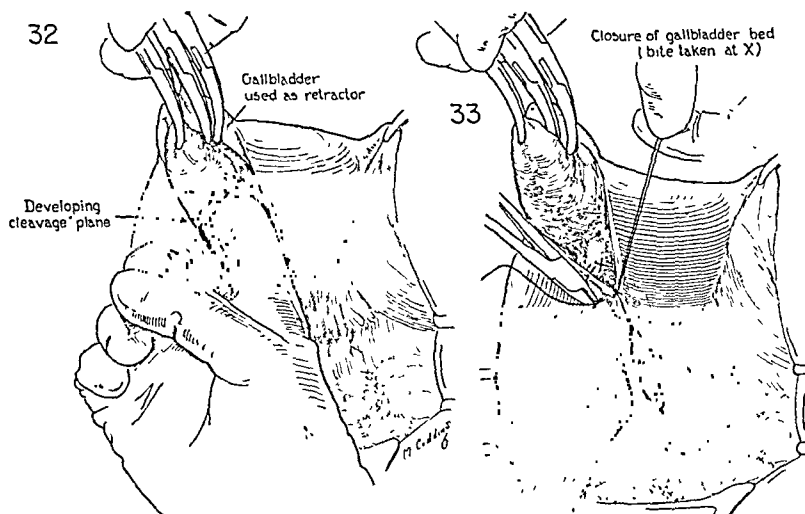
FIG. 32. An alternate method of removing the gall-bladder from its bed by developing a cleavage plane with the index finger. This may be a useful procedure, especially in acute cholecystitis.

FIG. 33. The gall-bladder bed is closed progressively as the gall-bladder is removed.
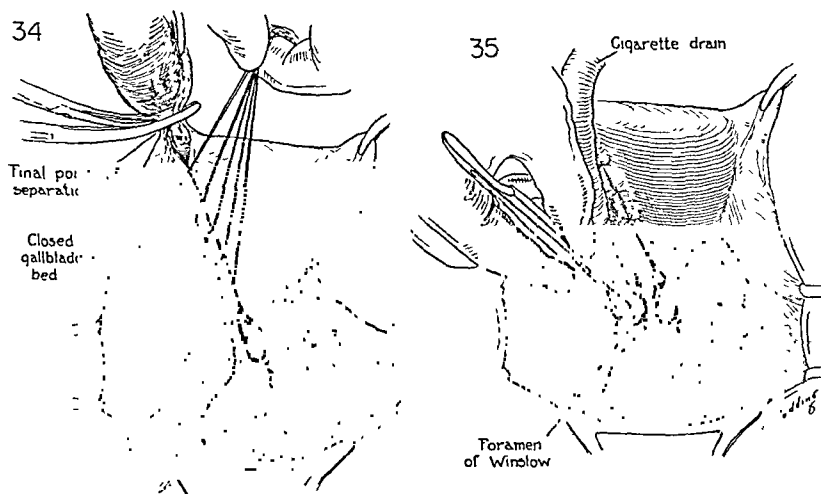
FIG. 34. The gall-bladder is retained as a retractor until its bed has been approximated with interrupted sutures.

FIG. 35. When indicated, a cigarette drain is inserted into the foramen of Winslow or down to the stump of the cystic duct.

Whether or not a cigarette drain should be inserted depends in our estimation on the acuteness of the inflammation in the gall-bladder, the amount of oozing from the field, and the adequacy of the peritonealization of the liver bed, as well as upon how secure and satisfactory the ligation of the cystic duct and cystic artery has been. We tend to insert a cigarette drain in fewer of these cases than formerly and have had no reason to regret this change in policy. The percentage of cholecystectomies not drained ranged from 20 per cent in 1935 to 46 per cent in 1938 with no increase in the mortality and no deaths from cholecystectomy in 1938 when almost one-half our cases were undrained. It is our belief that if proper hemostasis has been carried out with peritonealization of all surfaces, drains are unnecessary and may even be harmful. They add pain to the postoperative convalescence with resultant decrease in vital capacity; contribute to the general weakness of the wound; and give discomfort at the time of withdrawal.

But if drainage is to be employed, a cigarette drain with a gauze core which under no circumstances should extend beyond the rubber tissue is inserted down to the foramen of Winslow. The omentum is then brought up to the site of operation, the clamp is released from the round ligament, all gauze sponges are removed, and the liver is returned to its normal position. Some surgeons prefer to make a counter stab wound to the right of the major wound along the left costal margin to permit the cigarette drain to be brought out in this location. In making this stab wound care must be taken that no active bleeders are cut which will continue to bleed internally. When the surgeon is certain that there is no bleeding, the cigarette drain is anchored with silk to the skin. A routine closure is then effected.

After operation special attention is given to prophylaxis against pulmonary complications; the patient is placed in a semi-Fowler position, is moved from side to side frequently, and rebreathes carbon dioxide three to four times a day. The adhesive dressings and binder should not be tight enough to restrict breathing. Should pulmonary complications develop, the binder is further loosened. Since pulmonary embolus is an all too frequent catastrophe associated with biliary surgery, we have recently added several procedures to our routine care of patients in the hope of lessening its occurrence. The legs are raised to heart level immediately after operation and kept elevated for several days. Bicycle exercises are

encouraged starting on the first postoperative day. Women receive daily douches starting on the first postoperative day in an effort to overcome local venous stagnation, since the pelvic venous plexus is the source of many emboli. In addition vaginal hygiene may lower the incidence of such complications as cystitis.

Usually 2000 to 3000 c.c. of 5 per cent dextrose in isotonic saline is given intravenously during the first twenty-four hours after operation, the total being regulated to insure a urinary output of approximately 1000 c.c. daily. All intravenous fluids are given slowly. We prefer to administer fluids intravenously instead of subcutaneously in the great majority of instances, but in elderly patients or those with cardiac difficulty fluids are best administered subcutaneously to be taken up as needed.

TABLE I
OPERATIONS FOR CHOLELITHIASIS
Peter Bent Brigham Hospital 1934–1938

	Number	Average Age	Mortality Per Cent
Total operations*...................................	397	..	4
Cholecystectomy			
Chronic cholecystitis.............................	166	45	1
Acute cholecystitis...............................	67	51	3
Cholecystectomy and choledochostomy...............	136	54	5
Re-exploration of common duct......................	9	58	11
Cholecystostomy			
Acute cholecystitis...............................	19	55	21

* Gallstones were recovered in 94 per cent of all operations on the gall-bladder and common duct (exclusive of carcinoma and stricture).

Cigarette drains are removed within twenty-four to forty-eight hours unless there is bile leakage when the drain is not removed until a sinus tract has formed, usually five to seven days.

The average postoperative hospitalization for patients having cholecystectomy has been seventeen days. Our mortality in 166 cholecystectomies for chronic cholecystitis and cholelithiasis was 1 per cent, in sixty-seven for acute cholecystitis 3 per cent. (Table 1.)

Upon discharge patients are given instructions to follow a low fat diet and to avoid for several months those foods that proved upsetting before operation. Medical supervision is required for several months at least to insure the maximum relief from the surgical procedure.

COMMON DUCT STONE

One of the most common and serious mistakes in surgery of the gall-bladder and extrahepatic bile ducts is the surgeon's failure to search for and remove calculi located in or beyond the cystic duct. Sufficient statistics have been accumulated both from autopsy reports and clinical material to show that in approximately 20 per cent of all cases of cholelithiasis a stone is located in the intrahepatic or extrahepatic ducts exclusive of the cystic duct or gall-bladder. (Table ii.) From a study of the cases of cholelithiasis at the Peter

TABLE ii
COMMON DUCT STONE
Peter Bent Brigham Hospital 1934–1938

		Per Cent Mortality
Total operations for cholelithiasis	397	4
Cholecystectomy and choledochostomy	136	5
Choledochostomy	9	11
Incidence of choledochostomy*	36.5%	
Incidence of common duct stones in choledochostomy	51.0%	
Incidence of common duct stones in all cases†	18.5%	
Choledochostomy		
Stones recovered	74	9
Negative	71	0

* Annual incidence of choledochostomy varied from 51 per cent to 26 per cent.
† Annual incidence of common duct stones varied from 28 per cent to 12 per cent.

Bent Brigham Hospital over a five-year period, we find that the annual percentage of common ducts opened ranged from 51 to 26 per cent, that surprisingly enough the number of cases in which a common duct stone was recovered averaged 51 per cent of the ducts opened; that our annual incidence of common duct stone has varied between 28 and 12 per cent in direct proportion to the number of ducts opened. Since the number of common duct stones recovered will depend upon the number of ducts opened and explored, there is not much question that the incidence of choledochostomy should range between 40 and 50 per cent of operations for cholelithiasis. Our incidence of common duct stones recovered, and this does not include mud and debris, was 18.5 per cent of all cases operated upon for cholelithiasis. A stone located in the common duct is a serious

complication which demands consideration in the differential diagnosis of the patient with biliary disease, and a determined effort must be made in every case submitted to surgery for cholelithiasis to make certain that none is overlooked.

The diagnosis of common duct stone is usually made from a history of jaundice, colic, vomiting, chills and fever associated with a past history consistent with a diagnosis of cholelithiasis. More and more it is being recognized that stones will occur in the common duct without accompanying jaundice or pronounced colic. However, colic is present in at least 90 per cent of the cases of common duct stone at some time, and practically all cases give a past history consistent with biliary tract disease. Initial and persistent epigastric pain strongly suggests that a calculus is distending the cystic or common duct. The incidence of epigastric pain is higher in the presence of a common duct stone than in any other complication of cholelithiasis. Not only is the location of the pain suggestive, but frequent and repeated attacks of colic imply the passage of multiple stones through the cystic duct into the common duct. The location and frequency of the pain should be carefully noted in every case of suspected cholelithiasis because each may offer a valuable clue to the position of the calculi.

Although digestive disturbances are commonly associated with cholelithiasis and many patients have nausea with induced vomiting, we believe that pronounced involuntary vomiting gives evidence of a stone outside the gall-bladder within the cystic or common duct. This is derived from our experimental observation that while distention of the gall-bladder does not cause vomiting, distention of the common duct constantly produces spontaneous vomiting.[26,27] We noted that involuntary vomiting occurred in approximately 90 per cent of our patients with common duct stone. When vomiting is pronounced, we consider it suggestive evidence of a cystic or common duct stone, even in the absence of jaundice. A definite number of patients with common duct stones have such a marked weight loss as a result of nausea, intolerance to food, and even vomiting that, when associated with jaundice, a diagnosis of malignancy may be suspected.

While chills and fever have long been associated with common duct stone, they occur in only about one-third of our cases. However, a history of cholangitis with cholelithiasis definitely suggests that the common duct should be explored.

Jaundice was noted either on admission or in the history of 85 to 90 per cent of our cases. The surgeon should keep this in mind at the time of operation, because, unless the operative findings clearly contraindicate a diagnosis of common duct stone, a choledochostomy may be wise. It cannot be too strongly emphasized that common duct stones may be found in patients whose jaundice occurred years before operation. There is too great a tendency to disregard the diagnosis of common duct stone unless the patient is markedly jaundiced at the time of operation.

Since jaundice so often accompanies cholelithiasis and other disease of the biliary tract, it requires special consideration. The common causes of obstructive jaundice which are benefited by surgery are mechanical blocking of the biliary system, usually from acute or chronic inflammation associated with calculi, and tumor of the ducts or the head of the pancreas.[30] The importance of attempting to differentiate between these two lesions cannot be overestimated. In the presence of malignant obstruction of the common duct the jaundice will probably be progressive in type, and early operation is advisable; while in the case of common duct stone obstruction a more conservative treatment may be judicious, and operation may be delayed until the jaundice has subsided and the patient is in a better general condition. One of the most important diagnostic points in favor of common duct stone is a past history consistent with gall-bladder disease which is obtained in practically 100 per cent of these cases. In addition 80 to 90 per cent of the patients with common duct stone have colic. At least one-third of the patients have jaundice one or more times previous to the present illness. The mere presence of jaundice, therefore, has not so much differential diagnostic value as the history of intermittent or decreasing jaundice. While a marked loss of weight is more suggestive of malignancy than common duct stone, it is present in about one-fourth of the stone cases due no doubt to the fact that many of these patients have a persistent nausea and intolerance to food. Common duct stones occur nine times in women to once in men. This sex ratio, although not specific, may occasionally influence the physician in making a diagnosis. The physical examination also may be of definite value in making such a differential diagnosis. The patient with common duct stone may be tender in the right upper quadrant or epigastrium, but almost never has an enlarged gall-bladder. Courvoisier's law, therefore, is

fairly accurate and may be of considerable value in making a differential diagnosis.

Certain laboratory examinations aid considerably both in making a diagnosis and in determining the best time for surgery. In addition to the routine urine and blood examinations, determinations of the icteric index are taken on alternate days in the acute phases of the disease. If after several days the jaundice is decreasing, operation is postponed until the temperature, if elevated, returns to normal and the patient shows general physical improvement as well as a more normal laboratory chart. A more complicated but valuable laboratory procedure is a determination of blood diastase, for when pancreatitis is associated with common duct calculus surgery is delayed. For this reason we make a blood diastase determination at the time the patient with jaundice is admitted. When the patient is seen early, the diastase is frequently elevated, but a diastase determination is only of diagnostic value within forty-eight to seventy-two hours after the onset of jaundice since it usually falls to normal limits thereafter.[6] If the diastase is elevated, it would seem preferable to delay operation until it returns to normal, a minimum period of three or four days.

During the period of observation the bleeding and clotting times are determined in every case of jaundice regardless of the severity. A determination of the blood prothrombin level is usually unnecessary unless a definite bleeding tendency is encountered. However, it is true that the bleeding and clotting times, as determined by the usual methods, may be normal yet the patient have a prothrombin content so low that with the inevitable blood loss from the surgical operation, postoperative bleeding may occur. It is advisable, therefore, as a routine to give vitamin K supplemented by bile salts three times daily for four or five days previous to operation, the dosage depending upon the improvement in the bleeding and clotting times and the prothrombin level. In our experience, however, some patients become so nauseated that they cannot tolerate the administration of vitamin K by mouth; it must then be given by intraduodenal instillation. With the success of vitamin K in controlling hemorrhagic tendencies the administration of calcium and other anticoagulents is no longer indicated. Since an occasional patient, despite adequate doses of vitamin K, will continue to show a bleeding tendency, we believe that every severely jaundiced patient subjected to surgery

should be typed, and donors should be available at the time of operation and in the immediate postoperative period.

The same preoperative preparation as recommended under chronic cholecystitis is carried out. In addition, an attempt is made to obtain a glycogen reserve in the liver. This is best accomplished by encouraging the carbohydrate intake by means of a high carbohydrate, high vitamin diet. If the patient is unable to take carbohydrates by mouth because of nausea, vomiting, or general lack of appetite, 1000 to 2000 c.c. per day of 10 per cent dextrose may be given intravenously. Such concentrated solutions, however, must be given very slowly at the rate of about 500 c.c. per hour to avoid a diuretic effect.

Since these patients are on the average slightly older (54 years as compared to 45 years) and the mortality in these cases is about five times higher than in uncomplicated cholecystectomy, every detail of the preoperative preparation is carefully planned to bring each patient to operation in as good condition as possible. As previously stated, if the jaundice is subsiding, operation is delayed in the patient suspected of having a common duct stone until the icterus practically disappears. However, if the icterus remains constant or increases and the patient shows signs of cholangitis, as manifested by chills and fever, operation is carried out as soon as the fluid balance is properly adjusted.

Here again the anesthetic used depends upon whether or not the patient is jaundiced, since avertin is not given in the presence of jaundice. In elderly, poor-risk patients local infiltration of novocaine without adrenalin is satisfactory. In general the surgeon should use the type of anesthesia with which he is most familiar, as well as that which can be best given with the facilities available.

Before operation the surgeon should review the patient's history, especially in regard to the following points, and weigh well the possible need for choledochostomy.[8] Is the patient jaundiced or is there a history of jaundice? Has he had frequent attacks of gallstone colic or epigastric pain? Has there been pronounced involuntary vomiting? Is cholangitis associated with cholelithiasis? Have there been recurrent symptoms after cholecystectomy? With one or more of these findings present, the surgeon should realize before operation that exploration of the common duct may be necessary, but the operative findings will tip the balance.

As a routine in every operation on the biliary tract the common duct is thoroughly palpated, as described under cholecystectomy. But the decision to do or not to do a choledochostomy should not
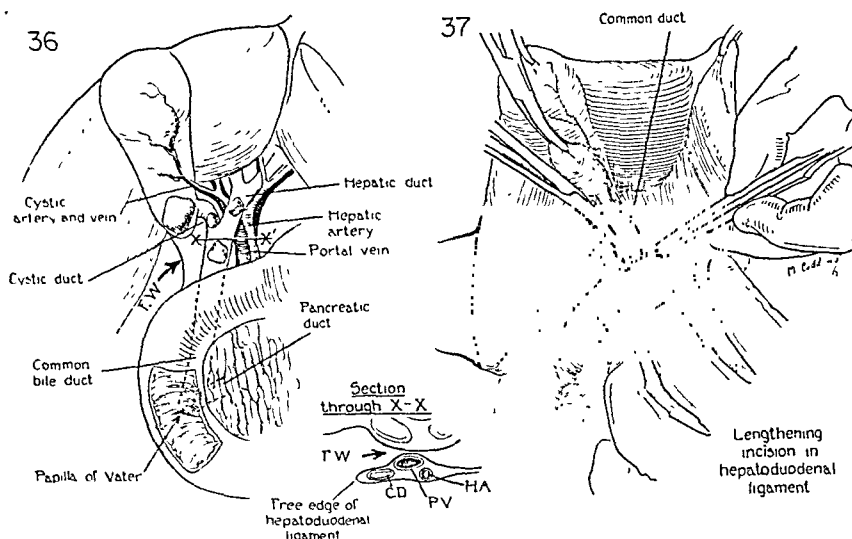
FIG. 36. Schematic drawing of the gall-bladder and extrahepatic ducts with adjacent structures showing the common locations of calculi. The cross section through x-x' shows the relationship of the common duct to other structures in the hepatoduodenal ligament.

FIG. 37. The hepatoduodenal ligament is divided to give adequate exposure of the common duct.

depend solely on palpation of the common duct. It is our impression that in general too much confidence is placed on the physical finding of whether or not the surgeon is able to palpate a stone within the duct. Frequently because of associated pancreatitis the head of the pancreas is considerably thickened, a finding which should influence the surgeon to do a choledochostomy. If upon palpation the gall-bladder is found to contain many small stones and the cystic duct is dilated so that one or more stones could conceivably pass, choledochostomy is compulsory. Conversely, if the surgeon finds an exceedingly small cystic duct and by palpation determines that the calculi within the gall-bladder are such that they could not possibly have passed through the cystic duct, choledochostomy is unnecessary even though many of the indications from the history are present. Many symptoms, such as involuntary vomiting and jaundice, may be produced by the inflammatory reaction about a stone located in the ampulla of the gall-bladder or cystic duct. The size of the

cystic duct, therefore, is of major importance. In the presence of a small, contracted, nonfunctioning gall-bladder, the common duct is always explored, for then it is usually impossible to determine

Palpation of common duct

Aspiration
of
common duct
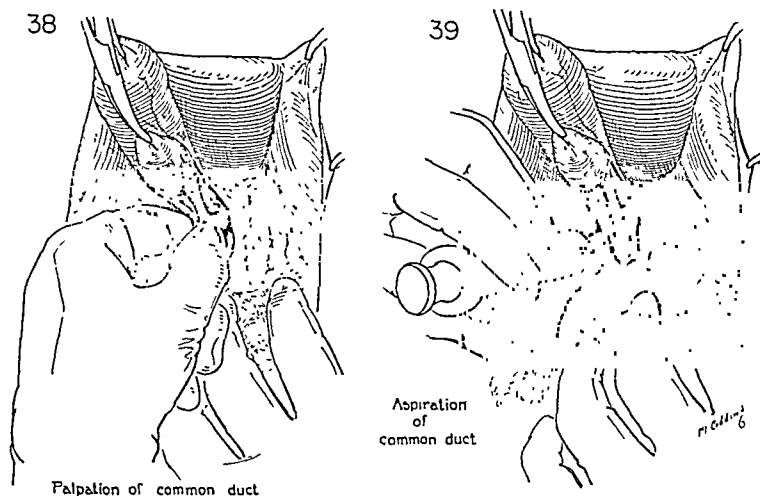
FIG. 38. The common duct and head of the pancreas are palpated by the left hand of the surgeon.

FIG. 39. The location of the common duct is verified by aspiration and the consistency of the bile is noted as a possible indication for subsequent exploration of the common duct.

whether or not a stone is present without exploration. Under these circumstances, the common duct is usually dilated.

Choledochostomy. The details of exposure of the region of the common duct are described under chronic cholecystitis. The common locations of gallstones are shown in Figure 36. The small inset (Fig. 36) shows the relationship of the common duct to the portal vein and hepatic artery. Since it is necessary by actual inspection and visualization to note not only the size of the common duct but its thickness and any other signs of inflammation present, the peritoneum over it is carefully incised, allowing adequate exposure from the cystic duct downward as well as up over the common hepatic duct for a short distance. (Fig. 37.) When this is done carefully, it is usually unnecessary to ligate any vessels. In the presence of jaundice the cystic duct should not be divided nor the gall-bladder removed until the surgeon is certain that any obstruction in the region of the papilla can be removed; the gall-bladder may be needed for a short-circuiting procedure. For this reason in the severely jaundiced patient clamps are not applied to the cystic ducts, and the blood

supply of the gall-bladder is not interfered with until the possibility of carcinoma of the head of the pancreas is eliminated.

The common duct can usually be identified by its color and the fact that a small blood vessel runs along the anterior surface. After the duct is freed, it is palpated to see whether any stones can be felt. (Fig. 38.) It is always excellent judgment to identify the common duct by aspiration of bile through a fine hypodermic needle. (Fig. 39.) In addition to confirming its location, information as to the consistency and thickness of the wall and the appearance of the bile is ascertained.

Visualization of the common duct by the injection of a radio-opaque substance, such as hippuran or lipiodol, has been advocated in recent years at this stage of the operation in order that negative explorations of the common duct may be avoided and a lower mortality may result.[18] We do not take cholangiograms routinely at this time and see no reason to change our attitude toward opening the common duct whenever there is the slightest question that a stone may be located there. During the past five years seventy-one choledochostomies were done without recovering a stone and without a death. Our deaths following choledochostomy have all been in patients in whom a common duct stone was recovered. From our experience, therefore, negative choledochostomy has not added to the general mortality.

After an adequate and satisfactory exposure of the common duct is obtained, traction sutures are applied to it at a convenient point, usually just below the junction of the common hepatic and cystic ducts. (Fig. 40.) Some prefer to explore the common duct through the dilated cystic duct, even incising its margin to permit the exploration in this fashion. To us this seems a difficult approach which does not permit as thorough exploration of the common duct as we believe advisable. Allis forceps should not be applied to the margins of the duct; gentle traction may be obtained more safely by silk sutures. The field is entirely walled off with moist gauze packs, and a glass suction tube is placed down to the foramen of Winslow to remove bile as it escapes at the time the duct is opened. With a small, sharp knife, an opening, usually about 1 cm. in length, is made parallel to the long axis of the duct, avoiding if possible the blood vessel commonly found on its anterior wall. (Fig. 41.) This opening must be large enough to admit a small scoop without traumatizing or tearing the duct. The color and consistency of the bile is noted. A metal

probe, such as a uterine sound, is then directed downward through the papilla of Vater into the duodenum. (Fig. 42.) Not only can the patency of the papilla be determined at this time but, with a finger
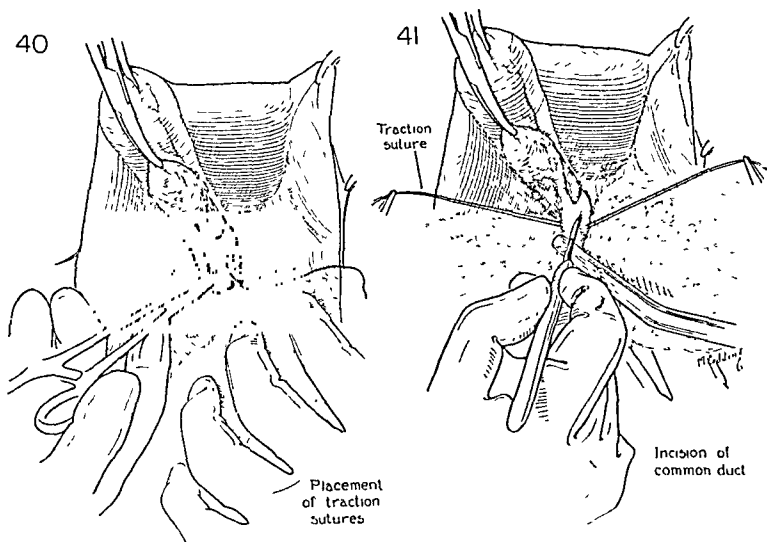
FIG. 40. Traction sutures are placed in the common duct below the point of entrance to the cystic duct in preparation for choledochostomy.
FIG. 41. The field is walled off with gauze packs, and an incision is made between the traction sutures into the common duct.

inserted in the foramen of Winslow to give counter-resistance as the metal probe passes, the surgeon may sense a grating of metal against stone. In this way a calculus which otherwise may be overlooked is found. The mere fact that the probe passes into the duodenum should not lull the surgeon into a false sense of security because frequently this is the case and yet calculi are either not impinged in the region of the ampulla or else are resting in a sacculation of the duct at this point. (Fig. 36.) A small pliant metal scoop is then directed downward into the region of the ampulla repeatedly and stones, if present, are removed. (Fig. 43.) A scoop 8 by 15 mm. with an easily moulded handle is entirely adequate; large scoops with rigid handles should not be used. The entire procedure is carried out very gently. The scooping is then directed upward into both hepatic ducts, for small calculi not uncommonly lurk in the larger intrahepatic bile ducts. (Fig. 44.) The scooping and subsequent steps of the exploration cannot be too thoroughly carried out, because autopsy findings, after even the most thorough explora-

42 Introduction of metal probe

43 Exploration of common duct with small scoop
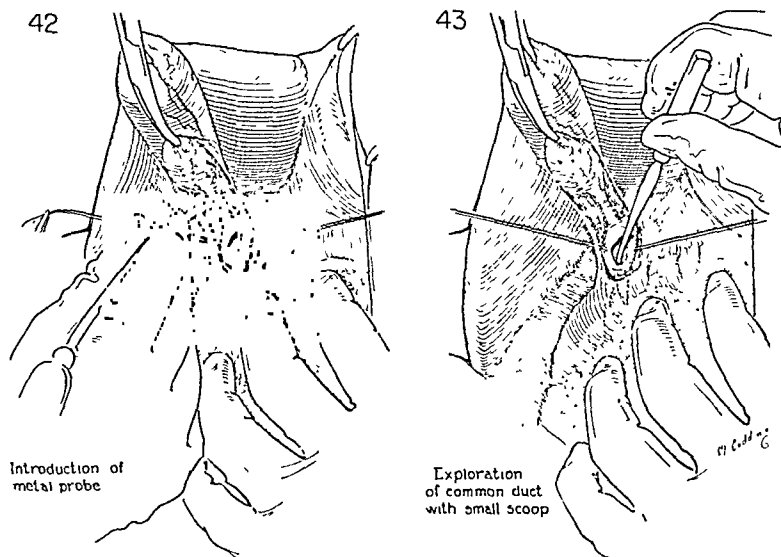
FIG. 42. The patency of the papilla of Vater is determined by the insertion of a uterine sound.

FIG. 43. A small scoop is introduced into the common duct down to the region of the papilla.

44 Exploration of hepatic ducts

45 Retrograde irrigation of hepatic ducts
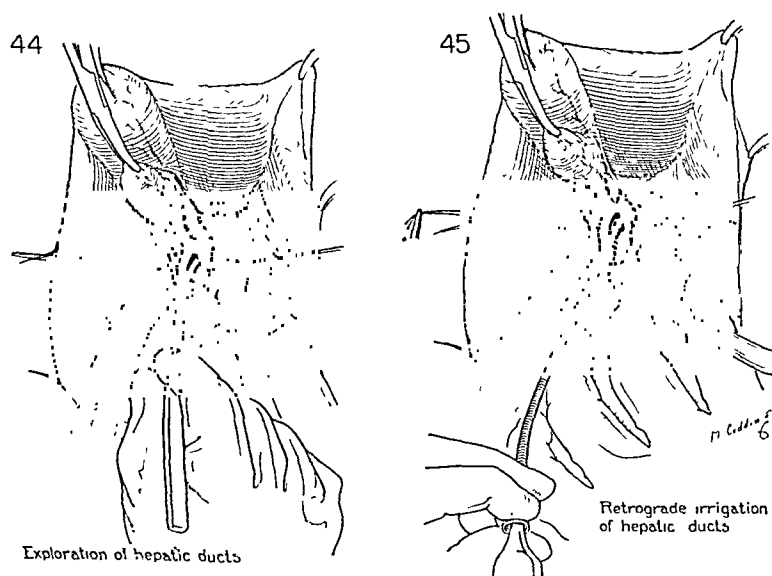
FIG. 44. The right and left hepatic ducts are thoroughly explored with a small scoop.

FIG. 45. The hepatic ducts are irrigated with saline through a silk woven catheter to dislodge any calculi.

tion, show in a distressingly high percentage of cases that small calculi are overlooked.

After the scooping has been carried out in such a fashion that the wall is not unduly injured and the opening in the common duct is

46   Determination of patency of lumen of papilla of Vater

Papilla of Vater

Olive tipped silk woven catheter

Injected solution

47

Dilated duodenum

48   Retrograde irrigation of common duct
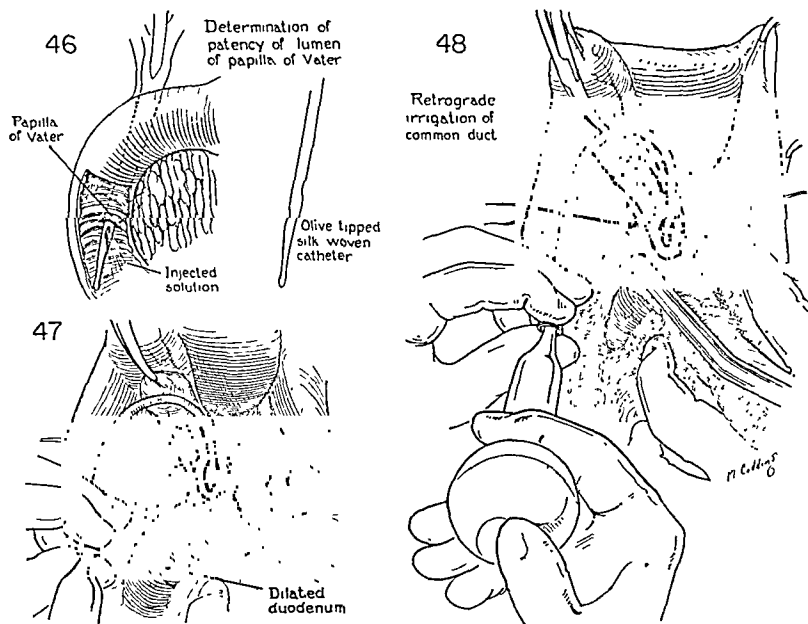
FIG. 46. Schematic drawing showing the olive tipped silk woven catheter within the lumen of the duodenum.

FIG. 47. A silk woven catheter has been passed through the papilla of Vater and its patency established by ballooning out the duodenum by the injection of saline.

FIG. 48. Saline is injected into the catheter as it is withdrawn to float up any debris or calculi in the common duct. The regurgitated material is aspirated through a glass suction tube.

not torn, a silk woven catheter, No. 12 or No. 14 French, is inserted toward the liver, and warm saline (110°F.) is injected as the catheter is withdrawn. (Fig. 45.) Under no circumstances should the surgeon guess at the temperature of the saline that he injects but should depend on thermometer reading. The stream of saline must be forceful as the catheter is withdrawn in an effort to dislodge and wash downward any calculi in the hepatic ducts. Meanwhile suction is maintained in the field to remove the escaping fluid. The catheter is then directed downward, and the surgeon notes the sudden increase in resistance encountered when it passes through the papilla of Vater into the duodenum. (Fig. 46.) Saline is injected and, if the

tip of the catheter is within the lumen of the duodenum, flows on to balloon out the duodenum. (Fig. 47.) We sometimes inject several hundred c.c. of saline through the catheter, since it offers an easy and convenient means of giving fluid. Saline is again injected vigorously with the asepto syringe as the catheter is withdrawn slowly to dislodge any calculi present. (Fig. 48.) Following this, the scooping is repeated.

Flexible silk woven catheters offer an ideal means for determining the patency of the papilla of Vater and permitting injection of saline to dislodge any calculi. Many surgeons prefer to pass bougies, starting with a No. 12 and increasing to a No. 14 or perhaps a No. 16 or larger, in an effort to dilate the papilla of Vater; while others recommend dilatation of the papilla of Vater almost to the size of the common duct by any method preferred, the contention in either case being that dilatation will permit better drainage of bile and the escape of overlooked common duct stones.[1] But it is our impression that a sphincter cannot be dilated all at one time and remain permanently enlarged. As a matter of fact forcible dilatation of the sphincter should result in immediate edema and subsequent scarring as a result of trauma to the papilla of Vater. For this reason a series of experiments, using various types of commonly accepted dilators was carried out in our clinic in animals.[4] These experiments proved to our satisfaction that in animals at least there resulted hemorrhage and inflammation in the acute stages following attempted dilatation of the papilla of Vater. The scarring, following this, resulted in a smaller opening than that which followed cholecystectomy alone. Therefore it would seem undesirable to attempt forcible dilatation of the papilla of Vater in patients undergoing cholecystectomy.

That dilatation of the papilla of Vater does not always leave a patulous lumen through which any overlooked calculi may escape can be shown by postoperative perfusion and ductal pressures.[29] We have found that on the first two days after operation the perfusion pressure was twice normal, showing the effect of the edema, no doubt from the attempted dilatation. The readings were taken at least six hours after the administration of morphine. In the presence of such pressures it is obvious that calculi could not pass, at least during the early stages. We have no evidence to support our theory that subsequent scarring and difficulty occur except that which can be gathered from our experimental findings. Control

experiments with a small catheter inserted into the cystic duct in a patient who did not have a choledochostomy and in whom the papilla of Vater was not disturbed were carried out. There resulted

49

50

Placement of anchoring
suture in open-end
common duct catheter

Introduction of
common duct
catheter
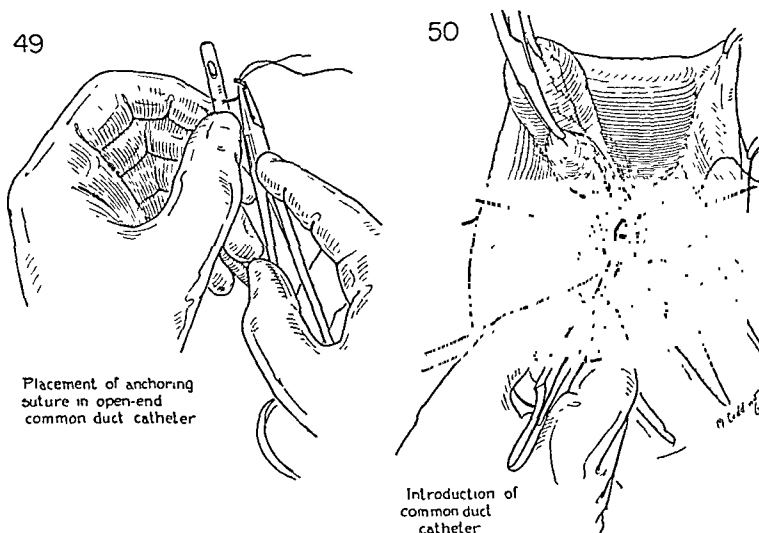
FIG. 49. A small bite is taken in the open-end catheter about 1 inch from the tip in preparation for anchoring it within the common duct.

FIG. 50. A small bite of common duct wall is taken with the anchoring suture. The catheter is directed up the hepatic duct.

only a slight elevation in perfusion pressure during the first few postoperative days, and the graph line was surprisingly flat. With this critical evidence to support our experimental findings, we feel rather strongly that the patency of the papilla of Vater should be determined by gentle methods and that under no circumstances should an effort be made to dilate it forcibly.

The problem of whether or not the common duct should be drained after it is explored has been debated for a long time. Some prefer to close the common duct primarily with fine silk sutures and to drain it through a small catheter inserted through the cystic duct;[25] others close the common duct primarily and insert a drain down to the point of closure. It is our custom to use a very small, open-end catheter (No. 16). The catheter is anchored with a silk suture about 2 to 3 cm. from its open end to the upper end of the common duct including just a small bite of the duct tissue (Figs. 49, 50 and 51); it is directed up the hepatic duct; and the suture is tied tight. By including only a small bite of the common duct and by tying the ligature tight it will slough through in a few days per-

mitting the catheter to be withdrawn. We do not recommend the use of T tubes or other types of angulated tubes, because they seem unnecessary and are more likely to traumatize the common duct

Closure of
common duct
around catheter

Testing
efficiency of
closure
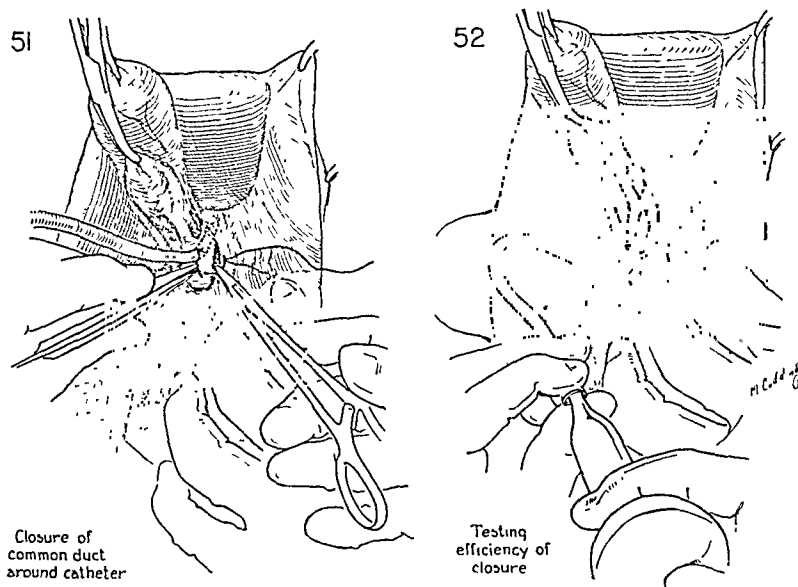
FIG. 51. The anchoring suture is securely tied, and the common duct is closed about the catheter.

FIG. 52. The efficiency of the common duct closure is tested by the injection of saline.

than a single catheter. However, if a T tube is used, the prolonged arms should be excised to cause as little trauma at the time of removal as possible. After the catheter has been anchored in place, the traction sutures are removed, and the common duct is closed with interrupted sutures of fine silk at the lower angle of the wound. (Fig. 51.) Saline is then injected to test the opening in the common duct. Obviously if the saline is injected under pressure, fluid will escape; but if the closure is relatively water tight, there will be no general escape of bile because the action of siphonage will prevent distention of the common duct after operation. (Fig. 52.) The subsequent steps of cholecystectomy are then carried out.

At this time in those patients having many stones in the common duct it is advisable to inject a radio-opaque substance, such as lipiodol, for a cholangiogram, since in the presence of multiple small stones in the common duct there is always the chance that some may be hidden within the hepatic radicles and be overlooked. It will also prove helpful in those instances where a probe or catheter does not

pass through the papilla into the duodenum by showing the contour of the stone and aiding materially in starting the subsequent steps of the surgical procedure. Similarly when a negative exploration of
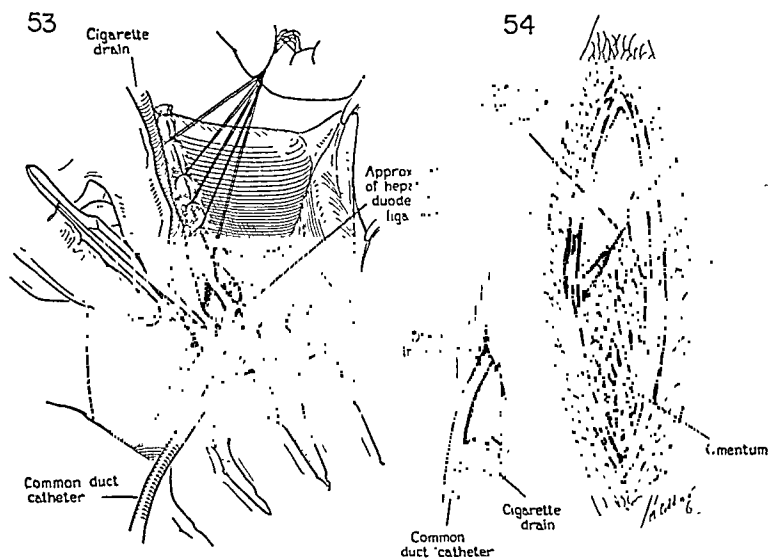
FIG. 53. The edges of the incised hepatoduodenal ligament may be approximated over the common duct. A cigarette drain is inserted into the foramen of Winslow.

FIG. 54. The catheter and cigarette drain are brought out unangulated through the wound or through a stab incision.

the common duct results, a cholangiogram may be useful to rule out stricture of any portion of the ducts as well as an overlooked calculus. It also should be considered in the case of a secondary choledochostomy.

The peritoneum about the common duct may be approximated with interrupted sutures. Whenever possible, the liver bed is peritonealized with interrupted sutures. (Fig. 53.) A final inspection is made to make certain that the common duct is not angulated or otherwise injured and that there is no evidence of bleeding. A cigarette drain is inserted down to the foramen of Winslow under the same circumstances as after simple cholecystectomy. After the table is levelled, the omentum is directed upward to cover the field of operation. The catheter and drains are brought out through the wound or through a stab wound to the right of the incision at a point where the drains will pass obliquely downward from the liver margin unangulated. (Fig. 54.)

Transduodenal Approach.   In rare instances when it is impossible to dislodge a calculus from the region of the papilla of Vater by careful and repeated manipulation a more drastic approach must
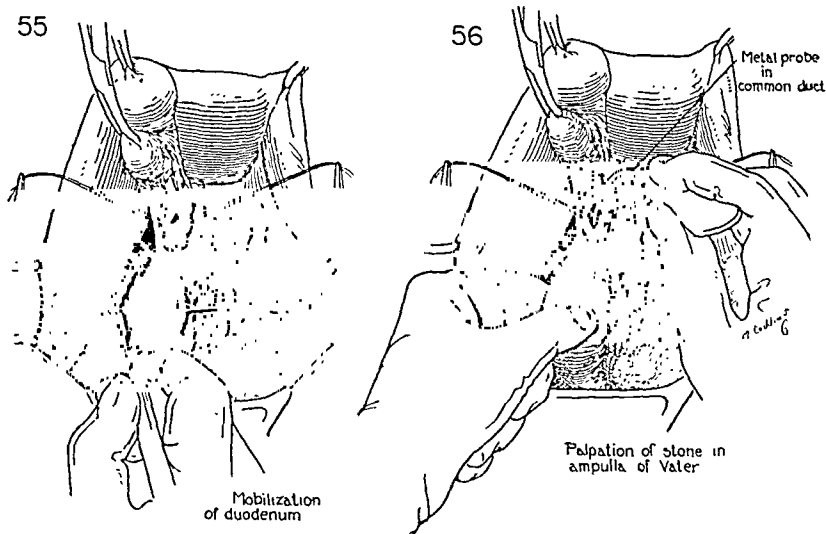
FIG. 55. The lateral peritoneal attachment of the second portion of the duodenum is incised in preparation for its mobilization.

FIG. 56. The position of the impacted calculus is more accurately determined after the duodenum has been mobilized.

be followed. Under such circumstances it is necessary to mobilize the duodenum and expose the papilla of Vater through a transduodenal approach. Since choledochostomy has already been done, an incision is now made in the lateral peritoneal attachment of the duodenum so that the second portion of the duodenum can be mobilized. (Fig. 55.) After the peritoneal attachment has been incised, either with a knife or scissors, blunt gauze dissection is used to sweep the duodenum medially. Occasionally this will expose the retroduodenal portion of the common duct, permitting more direct palpation, which, with the aid of a scoop in the common duct, may dislodge the calculus. (Fig. 56.) The scoop is passed down to the region of the ampulla, and its course is directed carefully with the index finger and thumb of the left hand of the surgeon. (Fig. 56.) Should this also prove unsuccessful, the anterior duodenal wall is incised, exposing the papilla of Vater.

By exerting gentle pressure on a uterine probe inserted in the common duct the surgeon can determine by palpation over the anterior wall of the duodenum the exact location of the papilla

of Vater. (Fig. 57.) With the duodenal wall held taut in Allis clamps
or by traction sutures of silk, an incision 3 to 4 cm. in length is
made over this area parallel to the long axis of the bowel. The field
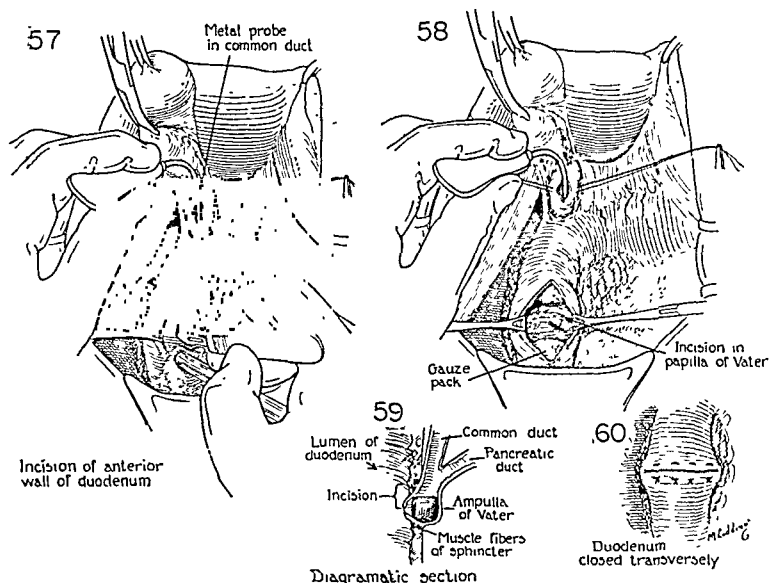
FIG. 57. The correct location for incision into the duodenum is determined
by means of a metal probe in the region of the papilla.

FIG. 58. The lumen of the duodenum has been walled off with a gauze
pack. A small incision is made directly into the papilla with the metal
sound in place.

FIG. 59. Cross-section of the papilla of Vater showing the impacted stone
and the extent of the proposed incision into the papilla.

FIG. 60. The duodenal wall is carefully closed in the direction opposite to
which the incision was made to prevent stenosis.

must be completely walled off by gauze sponges, and constant
suction must be maintained to avoid contamination by bile and
pancreatic juice. Small bits of gauze are then introduced upward
and downward within the lumen of the duodenum to prevent further
soiling. (Fig. 58.) Even at this point by direct palpation the calculus
may be dislodged. If this is still impossible, the probe is reintroduced
and directed firmly against the region of the papilla of Vater to
disclose the general direction of the duct so that a small incision
may be made directly parallel to it. (Fig. 58.) This incision enlarges
the papilla of Vater so that the calculus can be expressed. (Fig. 59.)
Any bleeding points from the incision into the papilla of Vater are
controlled by fine interrupted silk sutures. No effort is made to

reconstruct the papilla to its natural size, the opening being allowed to remain enlarged as a result of the incision.

Following removal of the calculus, bougies are introduced from above and repeated scooping and irrigation are carried out to make certain that no further calculi remain within the common duct. The small gauze sponges which plugged the duodenum are withdrawn, and the intestine is closed. To avoid constriction of the lumen of the bowel it is advisable to close the bowel in the opposite direction from which the incision was made. The duodenal wall is sutured with continuous silk on a straight milliner's needle, starting at the angle adjacent to one of the Allis clamps so that the knot is tied on the inside, and continuing as a Connell stitch over to the opposite clamp. Here again the knot is tied on the inside of the lumen so that the entire stitch may slough within the duodenum. The serosa is reinforced with a layer of interrupted silk Halsted mattress sutures placed with straight milliner's needles. (Fig. 60.) This closure must be water tight and secure to avoid the distressing complication of a duodenal fistula. A bougie is reintroduced into the common duct, and the duodenum is distended with saline to make certain that there is no leakage. A small common duct catheter is then directed into the opening of the common duct in the supra-duodenal portion, and the technique from this point on carried out as described under choledochostomy. A cigarette drain should be inserted down to the suture line in all cases to remain until there is no danger of a duodenal fistula.

A few special instruments drawn to scale in Figure 61 are recommended for cholecystectomy and choledochostomy. The silk woven catheter (Eynard No. 12 or 14) has a bulbous tip which expedites its passage through the papilla of Vater, and two eyes which permit direct injection of saline to prove the patency of the papilla and flushing of the ducts to wash out sand and mud as the catheter is withdrawn. The soft rubber catheter (Eynard No. 16) has an open end and an eye on one side. The small gall-bladder scoop, which because of its flexible handle should be a part of every surgeon's equipment for operations on the biliary tree, is in reality a Cushing pituitary scoop. These are available in various sizes. The Lower clamp which curves less sharply than a right-angle clamp, thereby permitting easier division of tissue between clamps serves the purpose of a right-angle clamp, and because of its length is more useful for cholecystectomy in our opinion.

Although we have not had a death in five years following a negative exploration of the common duct, the mortality has been 9 per cent when a stone was recovered from the common duct. With
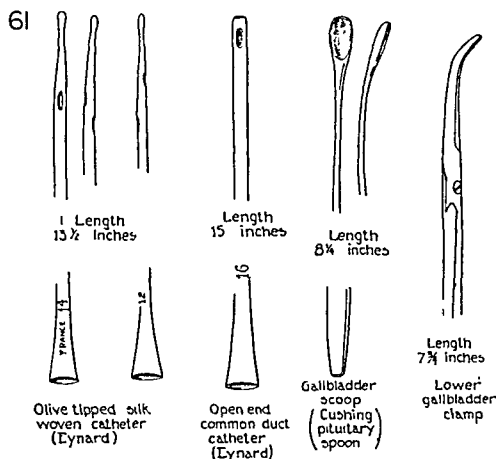
FIG. 61. Scale reproduction of the special instruments mentioned in the technique for cholecystectomy and choledochostomy.

such a high mortality compared to other operations on the biliary tract, it is important for the surgeon to keep in mind the seriousness of this operation and plan the postoperative care in great detail. The usual postoperative care as outlined under cholecystectomy is followed. But the patient having choledochostomy has also a draining catheter which must be watched closely. The surgeon is responsible for seeing to it that the catheter is adjusted to a constant drainage bottle immediately after the patient is returned to the recovery room and that it is not kinked or angulated. The amount of drainage from the common duct catheter is charted for each twenty-four hour period. When a small, open-end catheter is used, this usually averages 200 to 500 c.c. daily. If bile drainage ceases, the tube should be irrigated with sterile saline solution. The amount of drainage from the catheter frequently decreases from day to day and by the end of the tenth or twelfth day the catheter is removed. Usually there is no drainage twenty-four to forty-eight hours after its removal. Should the catheter be accidentally pulled out during the early postoperative period, no disastrous effects have resulted in our experience. We have at times followed the principle of keeping the drainage bottle elevated to direct more bile into the duodenum and to avoid too rapid decompression of the biliary tract. However, this

principle has not been strictly adhered to and we cannot present statistical evidence as to its effect on the postoperative course of the patient.

If the patient drains an excessive amount of bile or the amount of bile does not decrease, we attach the ordinary sterile intravenous saline outfit to the common duct drainage tube and note at what level above the common duct bubbles appear within the bottle, indicating that saline is entering the duodenum. This determines whether or not the papilla of Vater is patent and the resistance offered bile entering the duodenum. We do not use perfusion pressures routinely to determine when the catheter should be removed, but reserve the determination for the occasional patient in whom there is some question as to the patency of the papilla of Vater. If preferred, the common duct catheter may be clamped for one to two hours after meals, starting after the first week, to direct the bile into the duodenum. If the clamping of the tube produces distress, perfusion pressures should be taken to determine the patency of the papilla of Vater. We do not bother to clamp off the tube routinely because of the efficiency of the perfusion pressure in giving us the desired information. When high pressure readings are found, a cholangiogram is taken to determine whether there is a calculus or merely spasm of the sphincter of Oddi. It has not been our custom to do a routine postoperative cholangiogram because of the fever and discomfort it causes in some patients. A cholangiogram during the postoperative period may be indicated when a patient has recurrent pain or when profuse bile drainage persists beyond the usual interval. We believe that it should not be used routinely to determine the time when the catheter should be removed.

If it is shown by postoperative cholangiogram that a common duct stone was overlooked, an attempt may be made to dislodge it by irrigation through the common duct catheter, first with sterile saline which must be aspirated, and then with warm oil.[2] Occasionally the injection of oil by reducing the amount of friction will ease out the stone without further treatment. Nitroglycerine or atropine is always given before irrigation to insure relaxation of the papilla of Vater. Some surgeons advise the injection of ether in the belief that it will dissolve the stone, but we have not followed this suggestion.

In elderly patients who have been severely jaundiced there frequently develops a general apathy toward food following operation especially associated with loss of bile. In such instances the

patient is refed bile to the amount of several hundred cubic centimeters daily, usually by means of an intranasal Levine tube, although some patients will take bile mixed with tomato juice directly by mouth. These patients may be given 10 per cent dextrose by vein to increase the caloric intake.

If the patient has been deeply jaundiced, the administration of vitamin K is continued during the early postoperative period, and the icteric index is determined every four or five days. Stools are checked to make sure that bile is reaching the gastrointestinal tract. Five per cent dextrose in saline is given intravenously to maintain a urinary output of at least 1000 c.c. daily, but in the aged or those with cardiac disease subcutaneous administration is preferable. The average patient receives 1500 c.c. night and morning until fluids are taken by mouth. The amount of bile drainage must be taken into consideration in calculating the fluids necessary to be given the patient.

Cholangitis is not an uncommon complication of common duct stone. It is manifest by chills and not infrequently the classical intermittent fever of Charcot. In addition to the treatment described earlier under common duct stone, we have occasionally given patients with concomitant cholangitis sulfanilamide on the premise that cholangitis is associated with various organisms, some of which are known to be responsive to sulfanilamide therapy. But our results with this specific drug therapy have been variable. After operation in patients who have had cholangitis the common duct catheter is retained for a longer period than in the uncomplicated case. In no instance, however, has a common duct catheter been left in place longer than two or three weeks.

If a patient presents the symptoms of a common duct stone at a later date, an attempt may be made by suitable medication to encourage passage of the stone, the medication being directed toward relaxing the papilla of Vater and at the same time increasing the secretory pressure from the liver. The plan of treatment as outlined by Best and Hicken involves a three-day program. The first and third days .01 gr. of nitroglycerine is given by mouth three times a day; on the second day 0.1 gr. of atropine three times a day. In an effort to promote the flow of bile 2 or more ounces of epsom salts in warm water are given each morning, and each evening an ounce of cream or olive oil is given by mouth. During this three-day period a tablet of decholin is given four times a day to increase

the outflow of bile unless the patient is jaundiced. It is advisable
to examine the stools both during this period and for a few days
following to determine whether any stones have passed by way of
the gastrointestinal tract. We have had no proved case, however,
in which this regimen was effective.

ACUTE CHOLECYSTITIS WITH AND WITHOUT STONE

Although the classical description emphasizes the occurrence
of pain in the right upper quadrant with radiation to the angle of the
scapula accompanied by a tender mass in the right upper quadrant,
the symptomatology of acute cholecystitis may be quite varied.
Because of the extent of the acute inflammation, it would be expected
that the chief complaint of the majority of patients would be right
upper quadrant pain. However, this is not always the rule, for it
has been found that epigastric distress is nearly as common. Occa-
sionally the chief complaint is localized pain or tenderness in the
left upper quadrant or even in the right lower quadrant. Acute
distention of the gall-bladder or ducts as proved by our experi-
mental observations may explain the high incidence of epigastric
distress, especially in the early stages of acute cholecystitis. It should
be remembered also that not every patient having acute chole-
cystitis develops involuntary muscle spasm or a mass in the right
upper quadrant. As a matter of fact an analysis of 235 cases of
acute cholecystitis in the Peter Bent Brigham Hospital over a
period of twenty years showed that muscle spasm of the right upper
quadrant was present in 60 per cent at the time of admission and
a mass in the region of the gall-bladder in only 32 per cent.[5] This
may be explained by the fact that a thick-walled gall-bladder covered
by the liver does not bear any direct relation to the anterior parietal
peritoneum, and as a result the patient does not develop rigidity
in the right upper quadrant although severe epigastric pain as the
result of distention of the gall-bladder and cystic ducts is experienced.

Acute cholecystitis is one of the serious complications of gall-
bladder disease and requires careful judgment in its surgical treat-
ment. In our opinion cases of acute cholecystitis fall into one of
three clinical groups. The first group (approximately 40 per cent)
will respond promptly to the generally accepted conservative
measures, intravenous administration of fluid, sedatives, semi-
Fowler position, heat to the upper abdomen, and constant gastric
suction if distention is present. This therapy is aimed at treating

and improving the general condition of the patient. These patients will show such marked improvement under this regimen that surgery may be carried out safely within forty-eight hours of the time of hospital admission. A second and slightly larger group (50 per cent) responds more slowly. This is because the severity of the inflammatory reaction reduces the vital capacity and in spite of an adequate fluid balance and intake of food, the operative risk remains high. The fever, physical findings, and leucocytosis decrease gradually. Moreover, the surgeon may find an advanced degree of inflammation in the gall-bladder even when surgery has been *delayed several days after the physical and laboratory findings are* within normal limits, for though the general condition has been improved, the local pathology remains much the same. It is for this reason that we do not prolong the preoperative period. A third and fortunately smaller group (5 to 10 per cent) does not respond to the conservative treatment as outlined. The fever remains high, leucocytosis increases, and the physical findings in the right upper quadrant become more marked. It is apparent that these patients have a fulminating infection with impending or actual perforation; therefore immediate drainage of the gall-bladder is planned as soon as the fluid balance is corrected.

The explanation of these differing clinical conditions seems to us to result from the underlying etiologic mechanism. Thus what is usually classified as acute cholecystitis is chiefly a mechanical lesion in which obstruction to venous and lymphatic escape occurs, with resultant edema of the gall-bladder wall, which in turn further shifts its efficiency as a dialyzing membrane. Such cases, when seen early, present no additional risk. Unfortunately the surgeon has difficulty in distinguishing the simple mechanical acute cholecystitis from serious bacterial invasion. In the former group delay improves the condition, in the latter great delay may prove disastrous.

It has been our experience that patients with acute cholecystitis are in better condition for surgery if sufficient time is taken to correct the fluid balance as outlined in the conservative plan of treatment.[5] In our opinion a higher mortality will result in the hands of surgeons with limited experience if insufficient time is taken to prepare the patient properly for operation. The danger of a free perforation of the gall-bladder with a resultant general peritonitis is relatively uncommon in patients undergoing conservative preoperative treatment.

Upon admission to the hospital the patient with acute chole-cystitis is treated conservatively by the well standardized method of semi-Fowler position, morphine for pain, heat or cold to the right upper quadrant, and the intravenous administration of dextrose in isotonic saline. Since these patients may have considerable dis-tention, it may be advisable to place them on constant gastric suction according to the Wangensteen method. If there is a mass in the right upper quadrant, it is wise to determine accurately whether the mass is increasing or decreasing in size. The amount and extent of spasm and rigidity of the abdomen are determined several times daily. Each day a white count and differential are made and also a determination of the vital capacity, which may give valuable information as to the general improvement because so often patients have a marked limitation in their respiratory excursions as a result of the general tenderness in the right upper quadrant. It is the response to the general therapy which dictates the period of delay before surgery. If rapid improvement follows, surgery is performed once the fluid balance is corrected.

While in the great majority of our cases classical cholecystectomy as described in Figures 1 through 35 is carried out, not infrequently in the patient suffering from acute cholecystitis the surgeon en-counters a thick walled, tense, edematous gall-bladder with a stone impacted in the region of the ampulla and many adhesions attached to its under surface. Under such circumstances it is usually unwise to attempt a classical cholecystectomy because of the difficulty in isolating the region of the cystic duct and the possibility of acci-dental injury to the common duct or right hepatic artery. Rather than attempt to work blindly in this location by starting in a retrograde fashion, the operator should decide either upon a chole-cystostomy with simple drainage of the gall-bladder with the idea of carrying out a cholecystectomy later when the infection has sub-sided, a partial cholecystectomy, or if the general condition of the patient is good, removal of the gall-bladder from the fundus downward.

Cholecystectomy from the Fundus Downward. The field is well walled off with moist gauze pads, and the contents of the gall-bladder are aspirated through a trocar. A culture is taken of this aspirated fluid. As the trocar is withdrawn, a curved half-length clamp closes the opening to prevent soiling of the field. With the fundus of the gall-bladder retracted upward and outward and with

traction maintained on the round ligament as well, any adhesions to the under surface of the gall-bladder are separated by means of sharp and blunt dissection. (Fig. 62.) An incision is then made
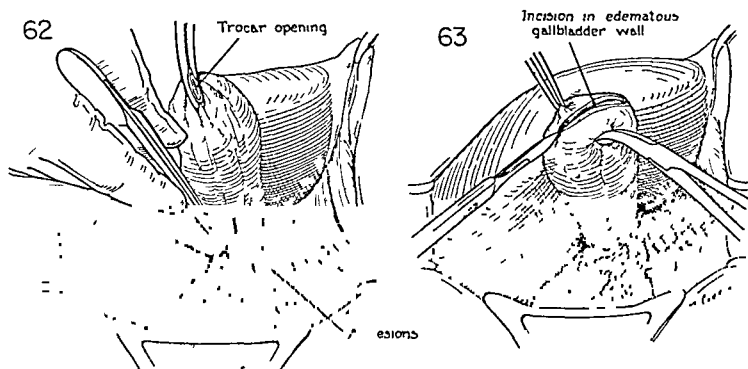
FIG. 62. Dense adhesions about an acute inflamed gall-bladder indicate removal of the gall-bladder from the fundus downward

FIG. 63. An incision is made in the edematous wall to develop a cleavage plane to facilitate enucleation of the gall-bladder from the fundus downward.
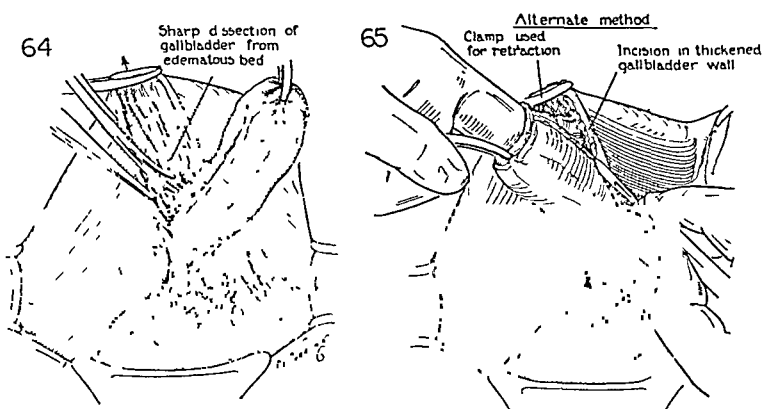
FIG. 64. Traction is maintained on the thickened cuff of gall-bladder bed at the liver margin while the gall-bladder is retracted medially to facilitate its dissection.

FIG. 65. As an alternate method the opening in the fundus may be enlarged and the index finger of the surgeon inserted to aid in the subsequent dissection and enucleation of the gall-bladder.

through the edematous serosa starting first around the fundus of the gall-bladder and extending downward on either side about 1 cm. from the liver substance. (Fig. 63.) Usually an avascular cleavage plane can be developed. The importance of incising the serosa early cannot be overemphasized because otherwise the acutely inflamed

gall-bladder may be torn from the liver, leaving an oozing raw liver surface. The cuff of thickened gall-bladder wall at the margin of the liver is held with a curved hemostat while the gall-bladder is further freed by means of sharp dissection. (Fig. 64.)
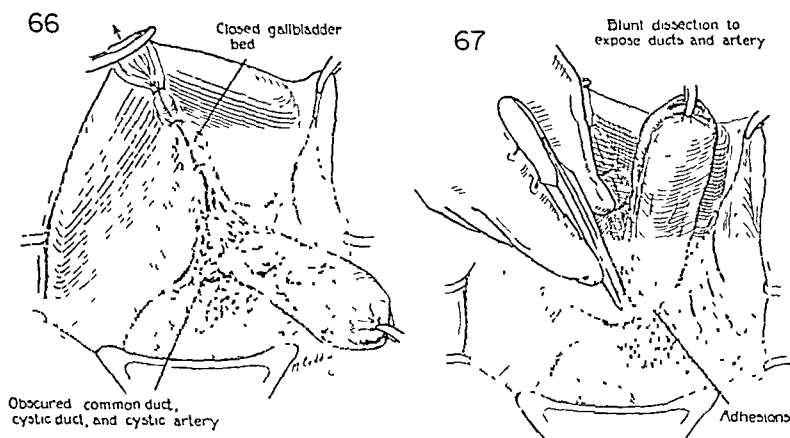
FIG. 66. The gall-bladder bed is closed to control bleeding before the cystic duct structures are isolated. Note that traction is maintained at the liver margin.

FIG. 67. Alternate blunt and sharp dissection is used to isolate the cystic duct and artery.

As an alternate method, since the contents of the gall-bladder have been aspirated and are frequently sterile, the opening in the fundus of the gall-bladder is enlarged, permitting the left index finger of the operator to be inserted to give counter-resistance and at the same time aid in dissecting the viscus within the previously developed cleavage plane. In other words, the gall-bladder can now be used much the same as a hernial sac when one is trying to separate it from its surrounding adhesions. (Fig. 65.)

The serosa is incised on each side alternately by sharp dissection until the region of the ampulla is encountered. Since there may be troublesome oozing because the cystic artery is still intact, all bleeding points are accurately clamped. The thickened gall-bladder is closed with interrupted silk sutures. The cuff of serosa at the margin of the liver is held by a curved half-length clamp throughout. (Fig. 66.) If the proper cleavage plane was not developed and the raw liver surface was exposed, an effort must be made to approximate these surfaces. Sutures of silk or fine atraumatic catgut on French needles are passed deep into the liver substance along the margin of the gall-bladder bed and are tied carefully to compress the bed, but not tear the liver substance. Great care must be taken in isolating

the ampulla of the gall-bladder from the common duct. Alternate sharp and gauze dissection is advisable until the majority of adhesions have been separated. (Fig. 67.)
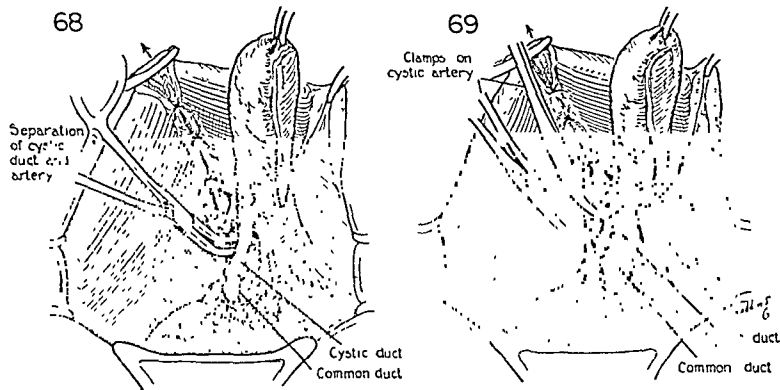
FIG. 68. The cystic duct is isolated by means of a Lower clamp from the common duct and cystic artery.

FIG. 69. The large cystic artery is clamped. It may be impossible to isolate it from all the surrounding inflammatory tissue.

After the ampulla of the gall-bladder has been carefully isolated from the region of the common duct, the cystic duct is isolated with the usual Lower clamp, very gingerly to avoid injury to the blood supply as well as to the common duct. (Fig. 68.) The cystic artery is next isolated with its accompanying indurated tissue, but an effort is not necessarily made to separate it as an individual vessel. The surgeon should keep in mind that when this procedure for cholecystectomy is necessary the cystic artery may be much larger than normal and the vessels may be in an anomalous location. The right hepatic artery should be definitely located lest it be clamped and divided. The cystic artery and adjacent tissues are then divided between a half-length and a right-angle clamp. (Fig. 69.) The artery is carefully ligated with a transfixing suture of fine silk. (Fig. 70.)

Attention is now directed toward the cystic and common ducts. It must be remembered that common duct stones are not infrequent in the presence of acute cholecystitis. During the past four years in the Peter Bent Brigham Hospital we have found it necessary to explore the common duct in 32 per cent of our acute cases with recovery of a common duct stone in one-half those explored or 16.5 per cent of all cases. A stone impacted in the cystic duct does not preclude a stone in the common duct, especially if the cystic duct is large and the stones in the gall-bladder are small.

If choledochostomy is to be included, the cystic duct is not divided until the common duct is explored. Otherwise it is divided and tied with a transfixing suture of fine silk. (Fig. 71.) After the
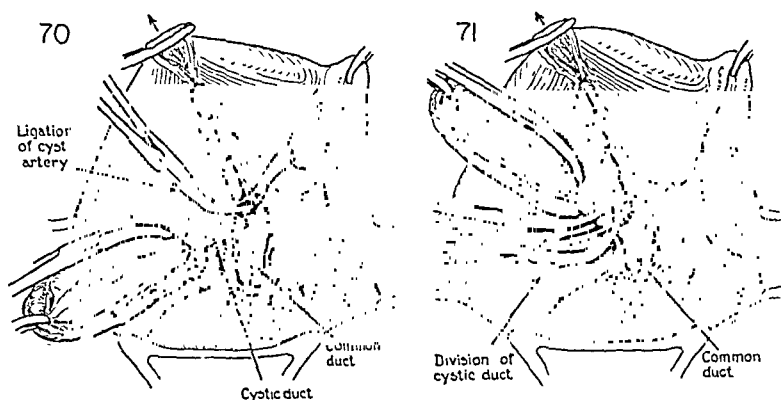
FIG. 70. The cystic artery is tied and all bleeding controlled before the cystic duct is divided because it may be necessary to explore the common duct.

FIG. 71. If exploration of the common duct is unnecessary, the cystic duct is divided and ligated in the usual fashion.

area has been repeatedly inspected for oozing, the clamp is removed from the liver margin, and a final suture is taken to close the gall-bladder bed at the liver margin. Since the local inflammation and technical difficulties have made this type of removal necessary, it is usually wise to insert a cigarette drain down to the region of the cystic duct. Should an area of raw liver surface be exposed, drainage is always indicated because of the danger of bile leakage. A routine closure follows.

Partial Cholecystectomy.   Instead of removing an acute, gangrenous gall-bladder from the fundus downward, some surgeons prefer to aspirate the contents of the gall-bladder and then partially resect it, leaving intact that portion adjacent to the liver.[10] This is a safe and satisfactory type of operation and offers as good a mortality and end result as cholecystectomy. It will, in a certain percentage of cases, save a patient from a second operation for complete removal of the gall-bladder. As far as can be determined there is no harmful effect from the remaining gall-bladder wall. This procedure may also be of value in those instances where the gall-bladder is so deeply buried in the liver that technical difficulties make cholecystectomy hazardous.

The field is exposed and the gall-bladder drained as for chole-cystectomy from the fundus downward. The gall-bladder wall is excised down to the region of the cystic duct which may or may not
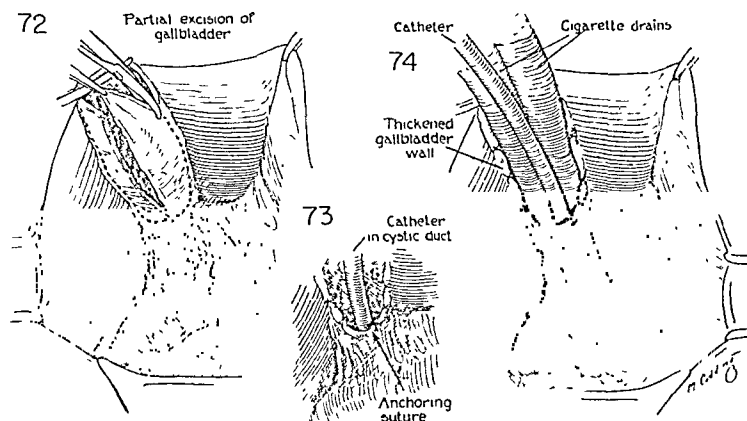
FIG. 72. The acutely inflamed gall-bladder wall is excised adjacent to the liver margin.

FIG. 73. If the cystic duct is not ligated, an attempt should be made to institute bile drainage by the insertion of a catheter.

FIG. 74. Following cauterization of the remaining gall-bladder wall and control of all bleeding points, several drains are placed in the remaining gall-bladder bed.

be ligated. (Fig. 72.) When the cystic duct is not ligated an attempt is made to insert a small catheter for bile drainage (Fig. 73), which is anchored in position with a transfixing suture. Any bleeding about the thickened, edematous gall-bladder wall is controlled either with a continuous stitch or interrupted sutures. An effort is made to destroy the remaining mucosa of the gall-bladder either by the application of actual cautery or some fixative, such as phenol or Carnoy's solution.

When this procedure is followed, it is implied that the common duct cannot be explored, and no effort is made to do so although a few patients will require reoperation. A postoperative cholangio-gram may be taken in those instances where a catheter has been inserted to ascertain whether a common duct stone is present, necessitating a second operation after the infection has subsided. Several cigarette drains are introduced down into the region of the cystic duct, and the omentum is brought up around these drains. A routine closure follows.

Cholecystostomy. Cholecystostomy, while no longer recognized as a routine method of treatment for the patient with cholelithiasis,

is occasionally a life-saving procedure in the presence of a fulminating, acute cholecystitis and was the procedure of choice on nineteen occasions during the past five years at the Peter Bent Brigham Hospital. Of the fifteen patients who recovered from cholecystostomy, five had a subsequent cholecystectomy during the same hospital stay. We look on this operation as a temporary procedure only, an invaluable aid in preparing the acutely ill patient for a subsequent cholecystectomy and thorough exploration of the biliary system after the infection has subsided and the patient is in better physical condition. Furthermore it is prudent to perform cholecystostomy in an elderly, poor-risk patient where removal of calculi is necessary because of repeated bouts of colic. On rare occasions in patients with obstruction of the common duct, long-standing jaundice, and a tendency toward hemorrhage which cannot be brought under control by vitamin K and transfusion, preliminary drainage of the biliary tract by means of cholecystostomy is in excellent judgment.

Usually the surgeon after a careful evaluation of the patient's condition realizes that cholecystostomy is the operation of choice. Under novocaine anesthesia a small incision is then made directly over the tender mass in the right upper quadrant so that its mid-portion will come directly over the maximum point of tenderness, usually the region of the fundus of the gall-bladder. Occasionally when technical difficulties or the severity of the infection involving the gall-bladder are not suspected previous to operation, it may be necessary to carry out this procedure through the usual right rectus incision.

No effort is made to dissect the adhesions from the under surface of the gall-bladder. The fundus of the gall-bladder is walled off with gauze in preparation for evacuating its contents. (Fig. 75.) An incision is made just through the serosa of the bulging fundus at the apex of the presenting dome of the gall-bladder, and a trocar is thrust through the thickened, edematous wall to remove as much of the liquid contents as possible. (Fig. 76.) Constant suction is maintained adjacent to the opening in the fundus as the trocar is withdrawn. A culture is taken routinely. The surgeon should inspect the aspirated fluid and estimate whether it is pus or bile-stained. It should be remembered that, even though the contents of the gall-bladder may appear purulent, they are frequently sterile, because the inflammatory reaction so often results from a mechan-

ical rather than a bacterial basis. After the trocar is withdrawn, the edematous wall is grasped in Allis clamps and the opening in the fundus is extended by scalpel or dissecting scissors. (Fig. 77.) The
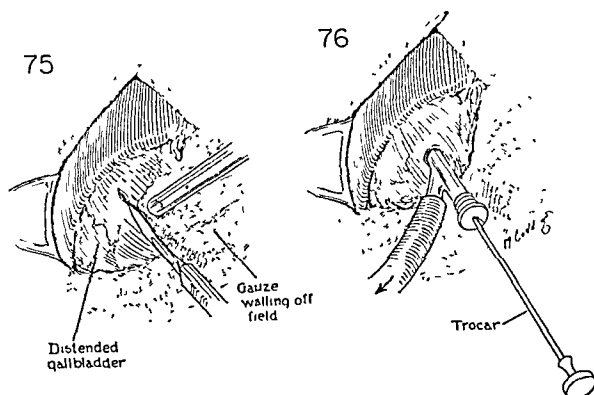
Fig. 75. The fundus of the acutely inflamed gall-bladder is walled off with gauze sponges, and an incision is made through the edematous wall in preparation for insertion of a trocar.

Fig. 76. The contents of the distended gall-bladder are aspirated through a trocar.

suction tube may be introduced into the lumen of the gall-bladder to remove any remaining liquid or grumous material. Since there is probably an impacted stone in the ampulla or cystic duct, an effort must be made to remove it to permit the escape of bile and drainage of the infected biliary ducts. A small, flexible gall-bladder scoop is introduced down to the region of the ampulla. (Fig. 78.) If the stones cannot be dislodged with a scoop, a fenestrated forceps may then be used. (Figs. 79 and 80.) If this too fails, no further attempt is made to remove the calculus, for frequently it is impossible to remove a stone held by the surrounding inflammation and the operation should not be needlessly prolonged.

The lumen of the gall-bladder is washed repeatedly with saline to flush out any remaining debris. A large rubber tube with a lumen at least 1 cm. in diameter is inserted down to the region of the ampulla and anchored to the fundus with an interrupted suture. (Fig. 81.) A purse-string suture of silk is placed in the fundal wall to close the gall-bladder snugly about the drainage tube. One or more sutures are taken to anchor the fundus to the peritoneum to prevent the possible escape of gall-bladder contents into the peritoneal cavity until the area is sealed off. (Fig. 82.) A routine

closure is then carried out. If the infection is severe or if an abscess is encountered and there has been soiling about the wound, a rubber tissue drain is inserted. After a sterile dressing is applied, the
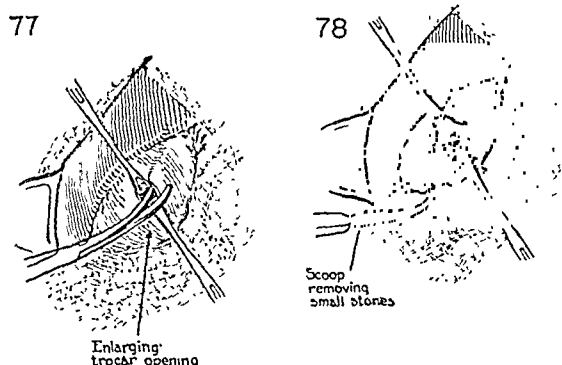
FIG. 77. The trocar opening in the fundus is enlarged to permit removal of calculi in the region of the ampulla.

FIG. 78. An attempt is made to remove calculi from the region of the ampulla by means of a scoop to relieve the cystic duct obstruction.
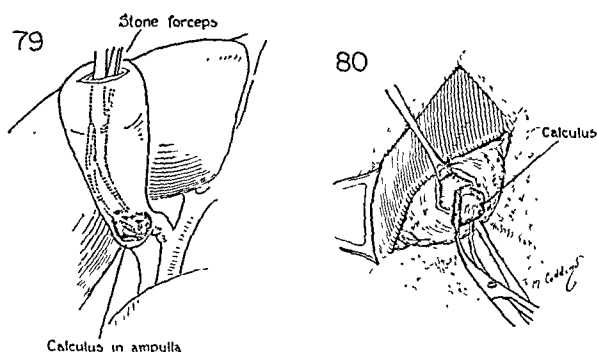
FIG. 79. Schematic drawing showing the common location of an impacted calculus in the ampulla.

FIG. 80. The removal of this impacted calculus may be facilitated by the use of a fenestrated stone forceps.

catheter is anchored to the skin with a suture or adhesive tape and is connected to a drainage bottle.

Following operation the patient is maintained in a semi-Fowler position, the fluid balance is corrected by the intravenous administration of fluids and the standard routine as described under chronic cholecystectomy is followed. Removal of the drainage tube will depend upon the amount of drainage from the catheter and the general condition of the patient as determined from the fever and

physical examination of the abdomen. Usually the catheter can be removed at the end of ten to fourteen days, and drainage from the sinus tract will cease shortly thereafter. While the tube is in place,
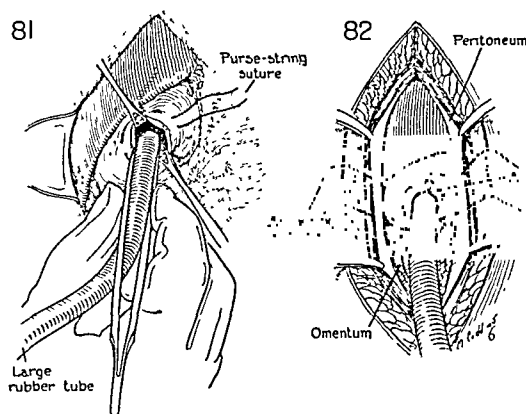
FIG. 81. Drainage is instituted by the use of a large soft rubber catheter inserted down to the region of the cystic duct. The opening in the gall-bladder is closed with a purse-string suture about the drainage tube.

FIG. 82. The drainage tube is anchored to the gall-bladder with suture A. The fundus of the gall-bladder, in turn, is anchored to the peritoneum.

hippuran or lipiodol may be injected, and a roentgenogram may be taken for evidence of overlooked calculi in the region of the ampulla, cystic duct, or common duct. If the general condition of the patient is good and the immediate postoperative recovery is uncomplicated, a subsequent cholecystectomy may be performed through the original wound two weeks after the original operation. However, if the patient is elderly or has been seriously ill, he is usually sent out of the hospital to return after three to four months for cholecystectomy. The only time that a secondary operation after cholecystostomy is not recommended is in the extremely poor-risk patient.

During the past five years there were 123 cases of acute cholecystitis at the Peter Bent Brigham Hospital subjected to operation, with a gross mortality of 7 per cent; in 115 there was also an associated cholelithiasis. (Table III.) Although the number of preoperative days of hospitalization averaged five regardless of the type of surgical procedure carried out, approximately 40 per cent of our cases of acute cholecystitis were operated upon within forty-eight hours. Cholecystectomy was performed in sixty-seven instances with

a mortality of 3 per cent. In addition, although it has often been implied that patients with acute cholecystitis are not so liable to common duct stone, the surgeon, after taking into consideration the various positive factors in the history of each patient, as previously mentioned, as well as the operative findings, felt justified in opening the common duct in thirty-seven cases or slightly more than one-third of those having cholecystectomy for acute cholecystitis. Common duct stones were recovered in 51 per cent of the choledochos-

TABLE III

ACUTE CHOLECYSTITIS

Peter Bent Brigham Hospital 1934–1938

	Average Days Pre-operation Hospitali-zation	Operation within 48 Hours	Per Cent Mortality
Total cases..............................	123	5	....	7
With cholelithiasis......................	115			
Without cholelithiasis..................	8			
Treatment				
Cholecystectomy.......................	67	5	32%	3
Cholecystectomy and choledochostomy....	37	5	40%	8
Cholecystostomy.......................	19	5	60%	21

Common duct stones recovered............................. 19

Incidence of common duct stone............................ 16.5%

Stones recovered at choledochostomy........................ 51.0%

tomies, or *16.5* per cent of the total acute cases. There was a general mortality of 8 per cent when common duct exploration was added to cholecystectomy; but in no instance was there a death following negative exploration of the common duct. Cholecystostomy was performed nineteen times with a mortality of 21 per cent. These nineteen were the poor risk patients with an advanced degree of inflammation, who probably would have presented a much higher mortality had more extensive surgery been attempted. In our opinion there was no instance where delay in operation was responsible for the increased mortality rate.

RESULTS OF OPERATIONS FOR CHOLELITHIASIS

· *Complications.* During the five-year period, 1934–1938, fifty-two, or 13 per cent, of our cases of cholelithiasis had postoperative complications. As would be expected, pulmonary complications were

the most common and occurred with greatest frequency in patients with acute cholecystitis or common duct stones. Surgery of the upper abdomen always carries a high incidence of pulmonary complications and this is especially true for procedures on the biliary system. Atelectasis was reported in seventeen patients, bronchopneumonia in five, a subphrenic abscess which required drainage in three, and two had a minor pulmonary embolus but recovered. It is apparent, therefore, that a pulmonary complication is the chief danger of the postoperative period and such measures as have been emphasized throughout under postoperative care should be instituted at once.

The second most common complication was wound infection, which occurred in sixteen instances, usually associated with acute cholecystitis. In two cases there was wound disruption. The interpretation of a wound infection no doubt varies with the individual surgeon and the severity of the infection, but we included those cases which showed induration although it was unnecessary to institute drainage.

Phlebitis occurred in six instances. This is a rather dreaded complication because it may be a forerunner of pulmonary embolus, which has been our most common cause of death. An attempt has been made, as stated in the postoperative care, to avoid this complication by elevating the legs immediately after operation, instituting bicycle exercises, and giving daily douches to women starting on the first postoperative day.

The balance of our complications consisted of two cases of hematoma, two of biliary fistula, and one of stricture of the common duct. Such complications may seem inevitable, but some are avoidable by the use of a precise technique in which the steps are carried out with adequate exposure.

Mortality. During the same period there were 397 operations for cholelithiasis, with a gross mortality of 4 per cent. Death occurred most frequently (eight cases), as shown in Table IV, in those cases where a common duct stone was recovered at operation. When a common duct stone was recovered, the cause of death was pulmonary embolus (four), gas bacillus infection (one), cardiac failure (one), acidosis (one) and hepatorenal syndrome (one).

We are loathe to make a diagnosis of hepatorenal syndrome, for in reviewing one hundred deaths occurring after surgery of the biliary tract we have failed to find more than a few which fill its requirements. We are in complete accord with the findings of

Touroff who believes that the surgeon should not classify deaths after surgery as hepatorenal syndrome unless all other causes have been ruled out and unless the characteristic changes involving the kidneys, liver, etc., are verified by autopsy and microscopic examination. Although some of these patients will present a terminal clinical

TABLE IV

DEATHS FOLLOWING OPERATIONS ON THE BILIARY SYSTEM*

Peter Bent Brigham Hospital 1934–1938

Age	Operation	Common Duct Stone	Path. Diagnosis	Days Post-operative	Cause of Death
72	Cholecystectomy and choledochostomy	Yes	Chronic	12	Pulmonary embolism†
62	Cholecystectomy and choledochostomy	Yes	Chronic	5	Gas bacillus†
62	Cholecystectomy and choledochostomy	Yes	Acute	21	Pulmonary embolism†
58	Cholecystectomy and choledochostomy ˋ	Yes	Chronic	9	Acidosis, hepatorenal syndrome(?)
59	Cholecystectomy and choledochostomy	Yes	Acute	20	Pulmonary embolism†
71	Cholecystectomy and choledochostomy	Yes	Chronic	1	Cardiac failure
72	Cholecystectomy and choledochostomy	Yes	Acute	13	Hepatorenal syndrome†
75	Choledochostomy	Yes	......	5	Pulmonary embolism
76	Cholecystostomy	...	Acute	9	Bronchopneumonia†
67	Cholecystostomy	....	Acute	6	Bronchopneumonia, cholangitis†
48	Cholecystostomy	....	Acute	1	Abscess of liver†
57	Cholecystostomy	.. .	Acute	36	Acute pancreatitis†
63	Partial cholecystectomy—Closure cholecystoduodenal fistula	....	Acute	1	Pulmonary embolism†
44	Cholecystectomy	....	Acute	8	Pulmonary embolism†
48	Cholecystectomy	....	Chronic	7	Bronchopneumonia
54	Cholecystectomy	....	Chronic	2	Pulmonary embolism
62	Average age				

* Exclusive of malignancy.

† Verified by autopsy.

history of rapid pulse, weakness, and progressive failure, which could be fitted into what is described in the literature as a typical picture of liver death, subsequent autopsy usually disclosed as the direct cause of death some unsuspected infection, such as subphrenic abscess, liver abscess, pancreatitis, peritonitis, concealed

hemorrhage or pulmonary embolus. We believe that one is not justified in designating a liver death without an adequate autopsy examination and that the so-called "liver death" or hepatorenal syndrome is rarely encountered. As a matter of record, in only one instance over this five-year period was the pathologist in complete accord with this diagnosis.

There were four deaths associated with cholecystostomy for acute cholecystitis, giving a mortality of 21 per cent. The mortality was high even though 60 per cent of these patients with acute chole- cystitis were operated upon within forty-eight hours after admission to the hospital when their fluid balance had been adjusted and it was apparent that their condition was not improving. We fail to see how any change in clinical treatment would lower this mortality rate because these patients died as a result of an overwhelming infection. In our opinion, the fact that the mortality was so great gives strength to the clinical judgment which prompted chole- cystostomy, for no doubt the mortality would have been higher had cholecystectomy been attempted.

There were only two deaths in the patients having cholecystec- tomy for chronic cholecystitis, giving an operative mortality of 1 per cent. As pointed out previously, this group was composed of younger, better risk patients and so gave a lower mortality rate. It is significant that during this five-year period, when a high percentage of these cases were operated upon by the surgical house staff that there were no deaths as the result of technical mistakes or hemorrhage.

Follow-Up. Upon discharge from the hospital, patients having the gall-bladder removed or drained are given instructions to follow a low fat diet and to avoid for several months those foods that proved upsetting before operation. Any tendency toward constipa- tion should be corrected, preferably by a morning saline cathartic. Each patient reports within a week for inspection of the wound and at regular intervals thereafter for a period of at least two years. If any patient has residual symptoms an attempt is made to over- come these by dietary measures and medication.

The records during this five-year period show that 212 patients were seen six months or longer after operation. No letter follow-ups were considered. Eleven complained of recurrent colic and severe distress; thirty-eight, including the eleven mentioned above, com- plained of some gas and indigestion. In addition there were eight

herniae, amounting to 4 per cent of the cases followed. The common duct was re-explored in nine instances, with recovery of common duct stones in six.

It is apparent, therefore, that although 94 per cent of the patients operated upon had calculi and the majority had colic, gas or mild indigestion was frequently reported after operation, clearly indicating the great importance of postoperative medical supervision.

INJURY TO THE EXTRAHEPATIC DUCTS

End-to-End Anastomosis. In rare instances the common duct may be accidentally divided. This is most likely to occur just below the junction of the hepatic and cystic ducts due to the technical errors described in Figures 16 and 21. The surgeon should be aware of an error at the time of operation and take steps to remedy it. Under such circumstances a direct end-to-end anastomosis is done with fine silk suture material. The peritoneum to the lateral wall of the duodenum should be divided, and an effort made to mobilize the duodenum because in all types of anastomosis of the common duct there must be absolutely no tension on the suture line. To this end mattress sutures of silk are taken in the hepatoduodenal and gastrohepatic ligaments anchoring the duodenum upward. (Figs. 83 and 84.) Clamps are not applied to the severed ends of the ducts. Should the edges be uneven or frayed, they are trimmed evenly to permit accurate approximation of the duct as in blood vessel suturing. Both ends of the duct are held in position with retention sutures of silk. (Fig. 84.) A layer of interrupted sutures is placed without entering the lumen to approximate the outer walls. Upon completion of the posterior layer (s_1), the mucous membrane is closed with interrupted silk sutures (B, C and D), the knot being tied on the inside of the lumen in each instance. Following this the common duct is exposed for a distance, preferably downward, to permit the opening of the duct as in choledochostomy and the introduction of a small open-end catheter. This is passed up beyond the suture line to insure an adequate lumen for the duct at the time the anterior layer of sutures is placed. (Fig. 85.) If the duct has been divided quite low, this opening may be made in the hepatic duct above the suture line, and the catheter may be directed downward. The mucous membrane of the common duct is closed over the catheter with interrupted silk sutures (s_3), the knot on the inside. A

second suture layer (s_4) is placed close to the original to reinforce the line of anastomosis. All the sutures taken in the duct must be accurately placed with small needles and fine silk and must include
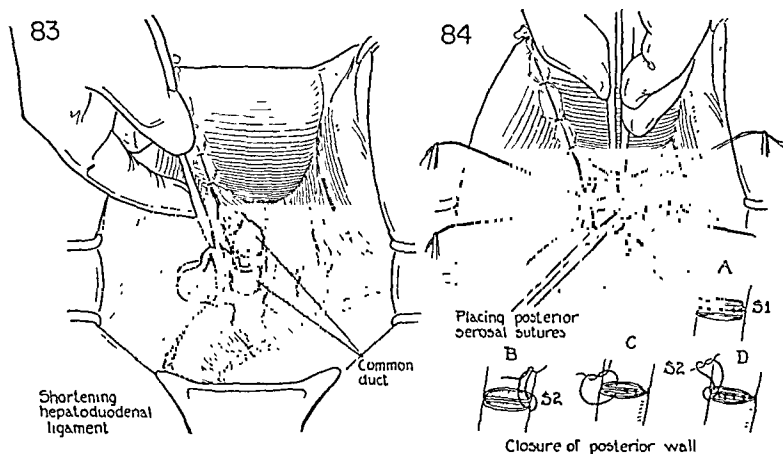
FIG. 83. The divided ends of the common duct are shown. The duodenum is drawn up toward the hilus of the liver by mattress sutures.

FIG. 84. The divided ends of the duct are held in approximation by traction sutures. The method of closure is schematically shown in A, B, C, and D.

only a minute bite of tissue to avoid stenosis. After the anastomosis has been completed, saline is injected into the catheter to make certain that there is no leakage about the suture line. (Fig. 86.) A final inspection is made to certify that there is no increased tension on the suture line. Should there be tension, additional anchoring sutures are taken in the adjacent ligamentous structure.

Strictures of the extrahepatic bile ducts may result from erosion of a calculus or prolonged inflammation, but the great majority are due to technical difficulties or surgical errors at the time of operation. The thoughtless surgeon, unable to control bleeding at the time of cholecystectomy, in desperation may clamp and ligate blindly with resultant inclusion of part or all of an extrahepatic bile duct. (Fig. 26.) Or he may fail to recognize the fact that the ampulla of the gall-bladder often lies parallel and adjacent to the common duct so that clamps applied roughly to the region of the ampulla in the early stages of cholecystectomy may also include a portion of the common duct, partially or completely dividing it. (Fig. 16.) Or since the cystic duct angulates the common duct sharply when the gall-bladder is retracted before the structures about the cystic duct are clearly defined, he may include the common

duct in his cystic duct ligation. (Fig. 21.) These technical mistakes the conscientious surgeon can overcome by following carefully each detail of the operative procedure and by resorting to chole-
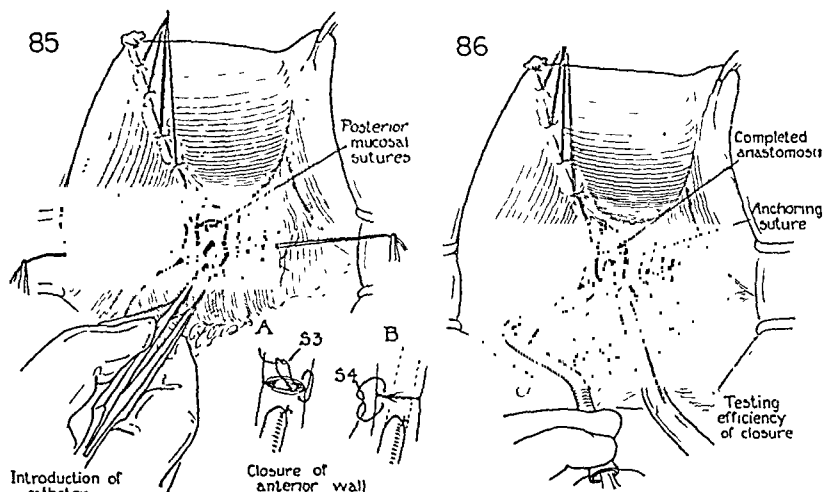
FIG. 85. An opening is made in the common duct below the point of anastomosis to permit the introduction of a catheter through the anastomosis. The method of closing the anterior wall of the common duct is shown in A and B.

FIG. 86. The security of the anastomosis is tested by the injection of saline.

cystostomy or removal of the gall-bladder from the fundus downward when the anatomy in the region of the cystic duct is distorted by extensive inflammation.

The symptoms of stricture depend on whether or not the duct was partially or completely ligated. If the duct was completely ligated, the patient may develop a severe and persistent jaundice immediately after operation. Stricture should also be considered in those patients developing profuse persistent biliary drainage during the postoperative period. Frequently the symptoms of stricture do not develop until months after operation. In such instances there has been a partial stenosis which remains asymptomatic until infection results from poor bile drainage. Then a painless jaundice of varying intensity, chills and fever, and all the signs of cholangitis may appear and recur intermittently. But since a common duct stone produces similar symptoms the surgeon must review the previous operative note. If there was difficulty with bleeding at the time of operation or technical difficulty in ligation of the cystic artery or cystic duct or profuse bile drainage, he should be suspicious of stricture.[16] Because these patients are usually jaundiced

the rigid preoperative preparation described under common duct stone for patients with jaundice is followed.

Heineke-Mikulicz Plastic. The most satisfactory method of handling a stricture cannot be determined until the involved area
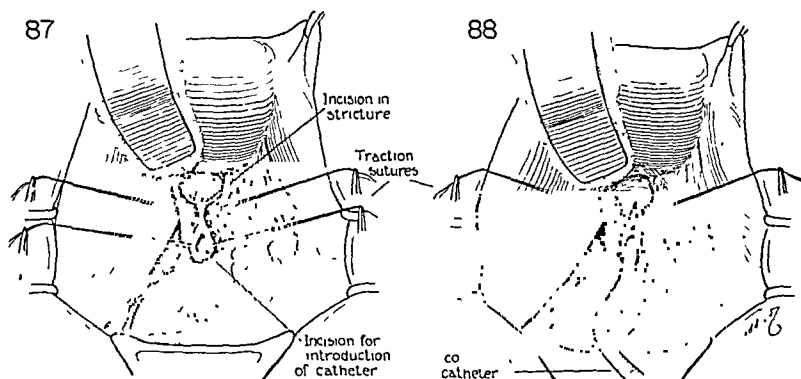
FIG. 87. This type of stricture of the common duct may be repaired by the principle of Heineke-Mikulicz.

FIG. 88. A small catheter is inserted beyond the point of the stricture.

has been thoroughly exposed. Occasionally a constricted area is found as the result of temporary application of clamps to the ducts. (Fig. 87.) In such instances the principle of Heineke-Mikulicz is used. Traction sutures are placed on either side of the isolated duct, and an opening is made either above or below the stricture depending upon the space available. An attempt is then made to pass a small probe or grooved director past the constriction. (Fig. 87.) With this metal director in place an incision is made throughout the constricted area. Traction sutures are placed in the cut edges of the ducts because it is necessary to sew up this opening in the opposite direction. An open-end catheter is passed through the previously made opening in the duct below the point of stricture, is directed beyond the constricted area, and is anchored in position with an interrupted silk suture. (Figs. 88 and 89.)

Before suturing the duct the duodenum must be thoroughly mobilized, so that there will be absolutely no tension on the suture line at the completion of the closure, in other words the distance gained must be more than the length of the vertical incision in the common duct. Frequently, however, because of a previous cholecystectomy, the duodenum is drawn up toward the hilus of the liver and mobilization of the duodenum is unnecessary. If it has been necessary to mobilize the duodenum, it is pulled upward and

anchored with interrupted silk mattress sutures which include a bit of the hepatoduodenal and gastrohepatic ligaments. After the duodenum has been freed and anchored in the desired position,
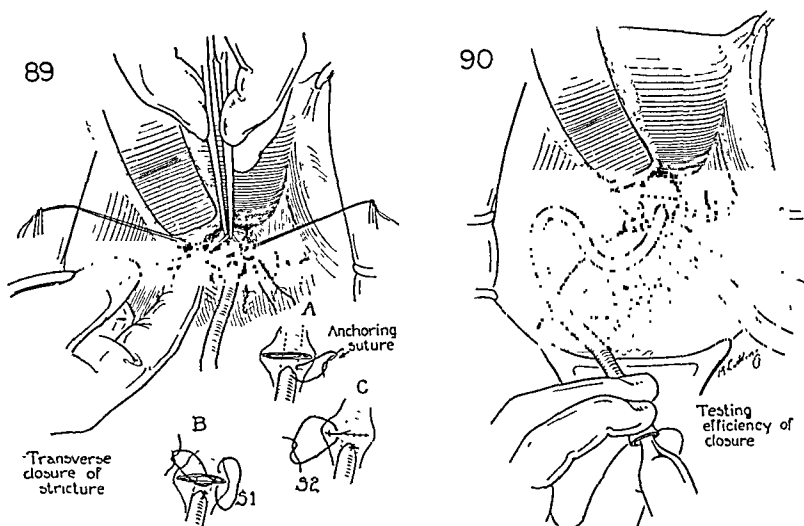
FIG. 89. The duodenum has been anchored upward to insure absence of tension on the suture line. The method of closure is illustrated in A, B, and C.

FIG. 90. The point of stricture is now the wide point of the common duct. Saline is injected to test the efficiency of the closure.

the opening in the duct at the point of stricture is approximated in the opposite direction with a fine silk suture.

The traction sutures are gently manipulated until the upper and lower lips of the opening approximate. (Fig. 89.) Interrupted silk sutures are taken including just the margin of the cut duct and are tied with the knot on the inside (s_1). A reinforcing layer of interrupted silk sutures (s_2) is then placed. The security of the suture line is finally tested by injecting saline through the catheter. (Fig. 90.) A final inspection is also made to insure that there is no tension on the suture line. The area is then covered with omentum, and a cigarette drain is inserted down to the anastomosis. A routine closure follows.

Anastomosis, Duct to Duodenum (with and without Tube). The surgeon is frequently faced with the difficult problem of finding the strictured area or ligated end of the common duct. When this is the case, he should not hesitate to follow his dissection high up into the hilus of the liver. The upper portion of the duct may be located by aspiration for bile with a fine hypodermic needle. Once its approxi-

mate location is determined, the dissection is carried well up into the hilus because it is better to attempt to isolate a small segment of duct and anastomose it directly to the duodenum than to institute tube
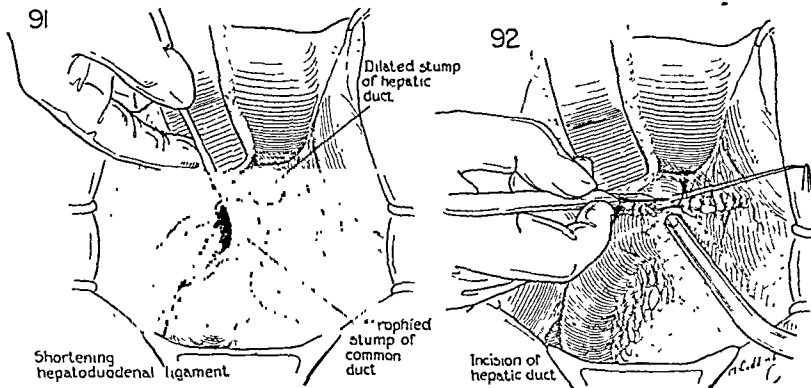
FIG. 91. The dilated stump of the hepatic duct has been located high in the hilus. The duodenum has been mobilized and is about to be anchored upward to permit a direct hepatoduodenal anastomosis.

FIG. 92. The dilated hepatic duct is held in position with traction sutures, and a small incision made into it in preparation for an anastomosis.

drainage and depend upon the formation of an external fistula which will have to be implanted subsequently. After the dissection has been carried well up toward the hilus of the liver and the dilated stump of duct is found, it is further isolated by sharp and blunt dissection. (Fig. 91.) Traction sutures of fine silk are taken in opposite sides of the duct, and with a small, sharp knife, an incision is made directly into the bulbous structure. (Fig. 92.) The initial incision should be small for it can be lengthened later or the anterior lip can be divided in a direction at right angles to the original incision as desired. This is done after the duodenum has been thoroughly anchored with mattress sutures up to a point where the anastomosis can be carried out without any tension on the suture line. (Fig. 92.) With the lumen of the duct held open by traction sutures of fine silk, the duct is anchored with interrupted silk sutures to the anterior duodenal wall at its superior margin. (Fig. 93.) These sutures (s_1) used in the anastomosis should not enter the lumen of the duodenum or the duct; any which do are replaced. It is usually necessary to take about four or five sutures for the posterior layer. A small incision is made directly into the duodenum close to the serosal sutures previously placed. (Fig. 94.) The field is well walled off during this time and constant suction is maintained adjacent

to the duodenal incision to prevent soiling from the escaped bile or duodenal juices. The mucous membrane is then closed using interrupted sutures (s_2 and s_3) with the knot always on the inside. A
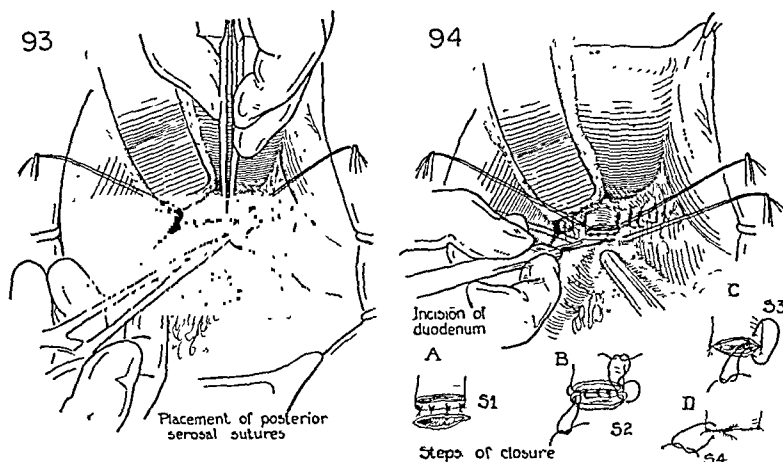
Fig. 93. The dilated hepatic duct is anchored to the superior margin of the duodenum.

Fig. 94. Interrupted sutures of fine silk are used to effect a two layer closure. The method of anastomosis is demonstrated in A, B, C and D.

reinforcing row of interrupted silk sutures (s_4) is placed close to the initial suture line to avoid turning in more tissue than necessary.

As an alternate method some surgeons prefer to use a rubber tube which, they believe, facilitates the anastomosis and prevents undue stenosis of the suture line. (Fig. 95.) A small rubber tube about 5 mm. in diameter, depending upon the size of the ducts, and about 6 to 8 cm. long is inserted for a short distance upward into the common duct and down into the duodenum after the two posterior layers of the anastomosis are completed. This is anchored to the duct with a fine silk suture which is tied very tight so that it will slough through in a few days. (Fig. 96.) The anterior wall is closed with two layers of interrupted silk sutures as shown in Fig. 94. If upon inspection there is tension on the suture line, the duodenum is further freed and reanchored with mattress sutures on each side of the site of anastomosis. (Fig. 97.)

Only the simpler types of anastomosis have been incorporated in this discussion. The direct anastomosis of the stump of the duct to the duodenum is probably the most satisfactory site of reanastomosis of the severed biliary tree to the gastrointestinal tract. But various other types have been recommended, and the surgeon operating

upon the patient with stricture should be familiar with all in order to select the one best suited for overcoming the technical difficulties encountered.
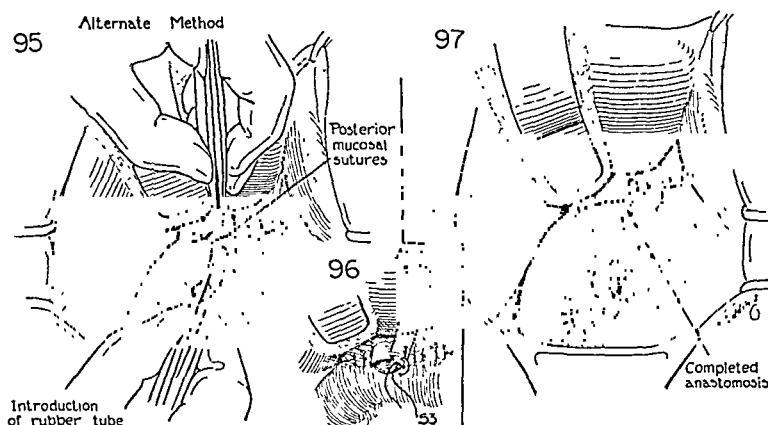
FIG. 95. An alternate method is the use of a small rubber tube to insure the patency of the anastomosis.

FIG. 96. The tube is anchored in place with a fine silk suture.

FIG. 97. The anastomosis has been completed and the duodenum has been fixed well up toward the hilus of the liver to avoid tension on the line of anastomosis.

CARCINOMA INVOLVING THE GALL-BLADDER AND EXTRAHEPATIC DUCTS

Within the past five years in the Peter Bent Brigham Hospital there were six cases of primary carcinoma of the gall-bladder, five of the ducts, and five of the head of the pancreas. In addition there was an occasional case in which metastatic glands from neoplasms of the stomach, breast, or sigmoid produced an obstructive jaundice due to extrinsic pressure on the extrahepatic bile ducts.

Carcinoma of the gall-bladder usually presents symptoms similar to those of chronic cholecystitis and cholelithiasis because of the high incidence of gallstones. Stones were recovered in 50 per cent of our cases and may have been present in others in which the neoplasm was so extensive that the gall-bladder was not opened. Stones, some suggest, are the causative factor of carcinoma of the gall-bladder due to the chronic irritation their presence creates. There is a common association with jaundice because of the presence of large metastatic glands in the portal fissures and possible involvement of the extrahepatic ducts by direct extension. Unlike chronic cholecystitis and cholelithiasis, however, the symptoms of carcinoma are progressive, with no tendency toward remission. Occasionally

the neoplasm may be so large that it produces a hard tender mass in the right upper quadrant. Usually, however, the diagnosis is not made before operation unless the mass is large and the patient has a concomitant history suggestive of malignant disease, such as weight loss, cachexia, etc. Although carcinoma of the gall-bladder occurs most frequently in the elderly (average 60 years), a very early carcinoma of the gall-bladder was found in a man 34 years of age having a cholecystectomy primarily for cholelithiasis. This suggests that early cholecystectomy should be recommended whenever the presence of gallstones is proved. In the majority of instances the extent of the neoplasm was so great that no more than biopsy of the lesion was attempted. Patients may show temporary response to x-ray treatment, but no curative effects will result.

Carcinoma of the bile ducts occurs usually in patients somewhat older (67 years) than those with carcinoma of the gall-bladder or of the head of the pancreas, and produces early intense jaundice due to complete obstruction of the common duct. It may or may not be accompanied by a past history of biliary tract disease. Diagnosis of carcinoma of the bile ducts cannot be made previous to operation. If the lesion occurs below the junction of the cystic and hepatic ducts, the gall-bladder may be enlarged and so may be used for a short circuiting operation to relieve the patient of the symptoms due to intense jaundice. During the past five years carcinoma of the bile duct was resected in one patient who did not recover.

Although our statistics show only five cases of carcinoma of the head of the pancreas during the last five years, this lesion occurs usually more often than carcinoma of the gall-bladder or the bile ducts. Along with common duct stone it comprises one of the two most common causes of obstructive jaundice. It is our belief that the physician can usually differentiate from the history and physical findings between jaundice-producing neoplasm and common duct stone with a fair degree of accuracy. In contrast to the symptoms of common duct stone described earlier, patients having neoplasm of the head of the pancreas rarely give a past history of gall-bladder disease or colic, though the majority have distress or pain of varying severity, usually in the epigastrium, right upper quadrant, or back. Weight loss is a primary diagnostic finding and occurs in almost 90 per cent of our patients, the average being 7 pounds per month. The presence of jaundice as such is not of particular diagnostic value unless it is continuous and increasing in severity. The sex ratio is

often of importance and may influence the physician making a diagnosis because of the high incidence of common duct stones in women and of neoplasm of the pancreas in men. As in neoplasms elsewhere, the patient with carcinoma of the head of the pancreas will give a history of increasing severity of all symptoms. The physical findings also may be of distinct value in making a diagnosis. A large, palpable gall-bladder in the presence of jaundice is presumptive evidence of neoplasm and was found in 55 per cent of our patients with carcinoma of the head of the pancreas. Enlargement of the liver is frequent in the presence of pancreatic neoplasm. However, an irregular hard mass was palpated in the epigastrium in only 9 per cent of our patients with carcinoma of the head of the pancreas.

Involvement of the gall-bladder and extrahepatic bile ducts with carcinoma offers little opportunity of early diagnosis or radical extirpation, but a patient may be offered palliative surgery with the relief of the obstructive jaundice. When the diagnosis of obstructive jaundice due to tumor is made, regardless of whether it is primary in the head of the pancreas or due to extrinsic pressure from metastatic glands, the physician should recommend surgical exploration. This attitude cannot be too strongly advocated because, regardless of how hopeless the situation may appear and how definite the diagnosis of malignancy may be, frequently the surgeon may find a calculus instead of neoplasm as the source of disease. Furthermore, in the cases of neoplasm a palliative surgical procedure may be carried out which will relieve the patient of the disagreeable symptoms associated with intense icterus. Occasionally a very small neoplasm, involving the head of the pancreas only, is found which may be resected successfully, as has been demonstrated by Whipple and his associates. The physician, therefore, must not take a hopeless attitude toward the surgical treatment of such patients. In our opinion, all should be offered the possible benefit of surgical exploration.

Since these patients are usually markedly jaundiced, attention must be directed toward combating a bleeding tendency as described under the treatment of jaundice. In addition at the time of operation it is advisable to have at least two blood donors available.

In view of the fact that these patients are generally in poor condition we prefer to carry out any operative procedure under local infiltration anesthesia (1 per cent novocaine without adrenalin).

Cholecystogastrostomy (Direct and Valvular Types). The abdomen is opened through a right rectus incision which is usually not carried as high over the xiphoid as is customary in cholecystectomy.
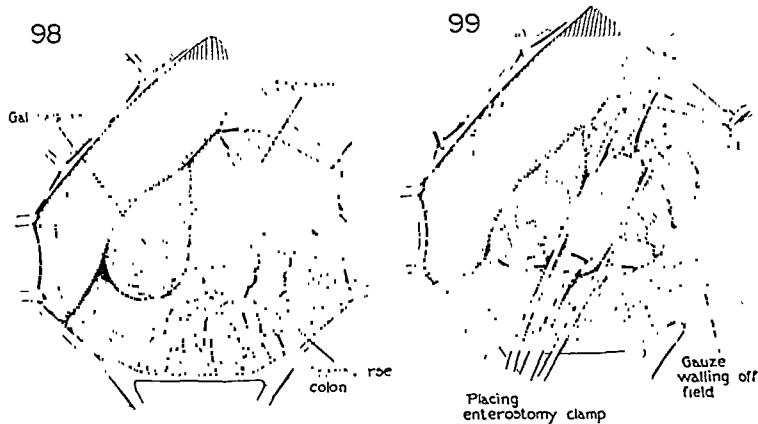
FIG. 98. The proximity of the elongated, dilated gall-bladder to the stomach and duodenum is illustrated.

FIG. 99. The anterior gastric wall adjacent to the gall-bladder is fixed with an enterostomy clamp in preparation for the anastomosis.

Every bleeding point, no matter how small, is tied to avoid if possible the danger of internal hemorrhage. The abdomen is thoroughly explored for evidence of metastasis, and the local cause of the distended gall-bladder is determined. The duct system is likewise inspected to rule out involvement by carcinoma, or extrinsic pressure from metastatic glands as is usually the case in carcinoma in the region of the head of the pancreas. It is also advisable to inspect and palpate the stomach for evidence of neoplastic involvement. What appears to be carcinoma from a malignant obstruction will occasionally prove to be a calculus. Even more trying are those cases in which there is no evidence of a mass that can be palpated. This is especially true in the very small tumors involving the lower end of the duct and the region of the papilla of Vater. If the gall-bladder is thin-walled and distended and offers no evidence of calculi, and if no calculi are palpable in the common duct, it is inadvisable to open the common duct. Since these patients are usually poor risks, it is better to decompress the biliary tree rather than attempt a more accurate diagnosis in the region of the ampulla by transduodenal approach. It has been our custom to anastomose the gall-bladder to either the stomach or the duodenum depending upon the

proximity of the fundus of the gall-bladder to these structures. If the lesion is small and the general condition of the patient permits, the problem arises of whether or not the original operation should be planned to permit resection of the head of the pancreas later.[24]
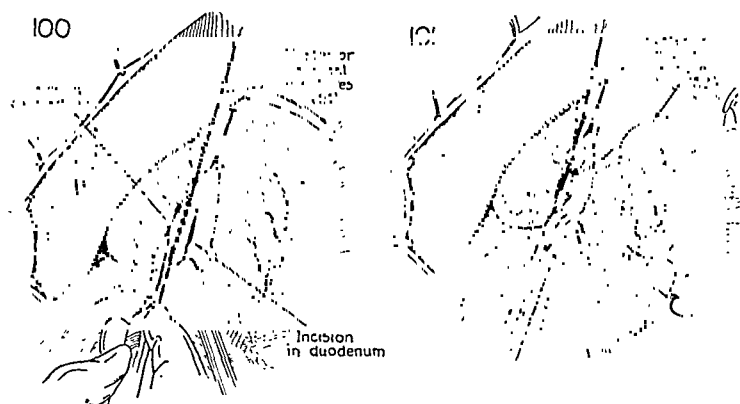
FIG. 100. A small opening is made into the stomach and gall-bladder adjacent to the suture line after the posterior layer of interrupted sutures is completed.

FIG. 101. After ligating any vessels of size the mucous membrane is closed with interrupted sutures of fine silk.

We have usually preferred to use the stomach for anastomosis because of its proximity to the enlarged gall-bladder (Fig. 98) although some prefer the small intestine[17] because it is more accessible and its wall approximates in thickness that of the gall-bladder. The anterior gastric wall near the region of the pylorus is grasped with Allis clamps between the lesser and greater curvature, and a straight enterostomy clamp without rubber covers is applied at the site of anastomosis. (Fig. 99.) This fixes the gastric wall to permit more accurate anastomosis of the fundus of the gall-bladder, to control bleeding, and to avoid contamination. The field is thoroughly walled off with gauze sponges. That portion of the gall-bladder which lends easiest approximation to the stomach is used in the anastomosis. Once the site is selected, a layer of interrupted sutures is taken to include the serosa of the gall-bladder and stomach for a distance of about 2.5 cm., the angle sutures being left long for traction. (Fig. 100.) Since the gall-bladder wall is very thin, great care must be exercised in placing the sutures that the wall is not torn. These sutures may be placed before the gall-bladder is aspirated, or if preferred, an opening may be made in the fundus of the gall-bladder with a large needle and the contents aspirated. Following this, an incision to permit a stoma about the size of the adult little finger is

made both in the stomach and in the gall-bladder. (Fig. 100.) The contents of the gall-bladder are aspirated with suction, every effort being made to avoid bile contamination within the peritoneal cavity.
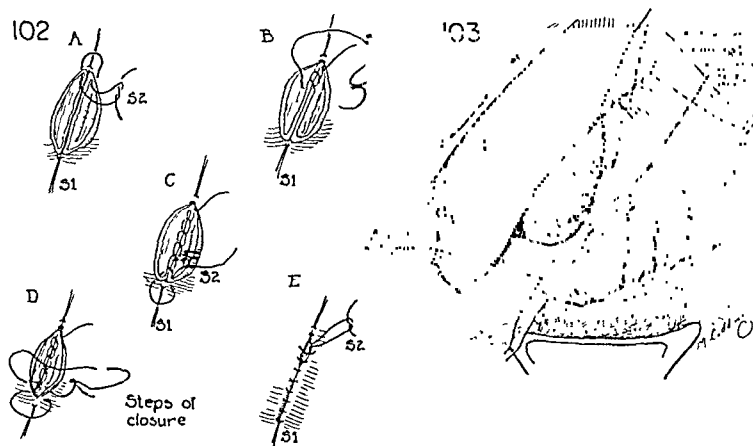
FIG. 102. Details of the anastomosis are schematically illustrated in the sketches A to E.

FIG. 103. Additional interrupted mattress sutures of silk are taken to insure the absence of tension at either angle of the anastomosis.

Although there are very few vessels in the fundus of the gall-bladder, sizable vessels, especially in the jaundiced patient, should be individually ligated. This applies to vessels in the gastric wall as well. The mucosa is then closed with a continuous silk suture, each stitch being placed close to the preceding one, since one of the functions of this suture is to avert hemorrhage. (Figs. 101 and 102A, B, and C.) The ordinary Connell type of stitch is used for the anterior surface. (Fig. 102D and E.) Following the completion of the mucosal suture, a second anterior layer of interrupted sutures is placed between the gall-bladder and stomach. (Fig. 103.) It is advisable to reanchor the stomach to the gall-bladder with several interrupted sutures (s_3) beyond the angles. In addition it is occasionally advisable to anchor the stomach to the region of the round ligament to remove tension on the anastomosis. Whether or not a gastroenterostomy is done will depend somewhat on the size of the lesion and on whether the surgeon plans to attempt a resection of the head of the pancreas later.

Recently we have used a valvular type of cholecystogastrostomy instead of the direct anastomosis previously described. Although the direct type of anastomosis has been moderately effective as a palliative procedure, it has been found by Whipple to result in cholangitis and hepatitis. For this reason various types of anastomoses have

been proposed in an effort to avoid ascending biliary infection. It is surprising that, in the absence of function of the valve-like mechanism of the sphincter of Oddi, a higher incidence of cholangitis and
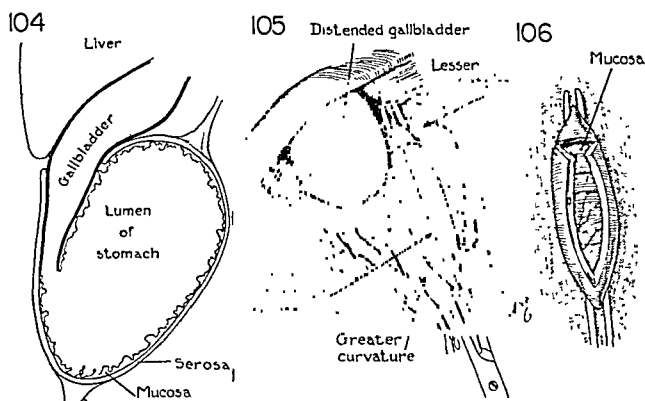
FIG. 104. Schematic drawing to illustrate how the valve-like effect is obtained by implanting the gall-bladder obliquely within the wall of the stomach.*

FIG. 105. A T-shaped incision is made through the seromuscular coat from lesser to greater curvature of the stomach in the region of the pylorus.

FIG. 106. The transverse incision near the greater curvature is made large enough to avoid constriction of the gall-bladder after implantation.

evidence of infection do not result when the biliary tree is anastomosed to the gastrointestinal tract. The lack of the valve-control permits an interchange of contents between the gall-bladder and the gastrointestinal tract which encourages mild infection that does not ordinarily produce clinical symptoms but yet may be of a very serious nature.

In an effort to avoid at least gross interchange between the contents of the gastrointestinal tract and the biliary passages, we have devised a valvular type of cholecystogastrostomy following a technique developed by animal experimentation. It was found that the gallbladder could be implanted for a distance between the seromuscular coat and the mucous membrane of the stomach and retain its viability, giving valve-like protection and preventing the contents of the stomach from entering the gall-bladder. Experimentally there was less dilatation of the biliary system following the valvular type of anastomosis than following the direct type.[28] Furthermore it was found that if the fundus of the gall-bladder was

* Figs. 104–114 inclusive are reproduced by the courtesy of Surgery, Gynecology & Obstetrics.

pulled into the lumen of the stomach, stenosis might result, and that a direct anastomosis must be made between the fundus of the gall-bladder and the mucous membrane of the stomach. We believe that
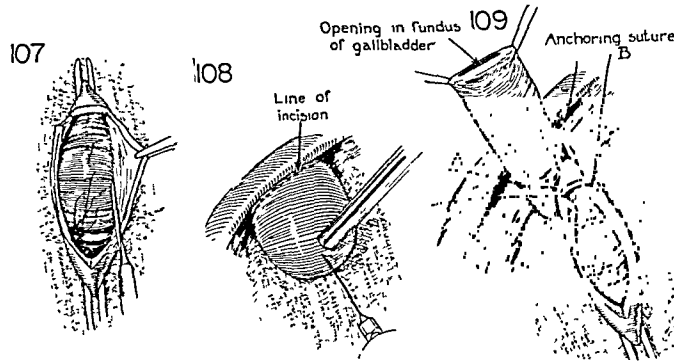
FIG. 107. The seromuscular flaps are developed on either side to give sufficient space for implantation of the gall-bladder.

FIG. 108. If the gall-bladder is not elongated sufficiently to permit implantation, an incision is made into the serosa to permit its possible detachment from the liver bed.

FIG. 109. The lesser curvature of the stomach is anchored deep to the under surface of the gall-bladder with interrupted silk sutures.

the valvular type of cholecystogastrostomy instead of a complicated type of anastomosis with the small intestine may be beneficially used. Since the gall-bladder in the presence of obstructive jaundice is usually dilated, it is sufficiently elongated to permit implantation in the gastric wall for at least 2.5 to 3 cm. (Fig. 104.) It may be safely detached from the liver bed without interference with its blood supply. That detachment of the region of the fundus of the gall-bladder is a safe and satisfactory procedure has been verified by us, both experimentally and clinically.

A portion of the anterior gastric wall which lies in close proximity to the gall-bladder is grasped near the greater curvature with Allis clamps, and a straight enterostomy clamp without rubber covers is applied to fix the gastric wall, control bleeding, and avoid contamination when the mucous membrane of the stomach wall is divided. (Fig. 105.) A T type incision is made to avoid constriction of the gall-bladder as it enters the gastric wall. This transverse incision should be 2 cm. long. (Fig. 106.) It is usually necessary to ligate a few vessels coming from the lesser curvature in this location. The seromuscular flaps are then dissected by sharp and blunt dissection from the underlying mucosa until easy implantation of the gall-bladder is permissible. (Fig. 107.)

The gall-bladder is now prepared for anastomosis. The contents are aspirated with a syringe or trocar through the fundus, at a point which has been selected for the anastomosis. (Fig. 108.) If the gall-
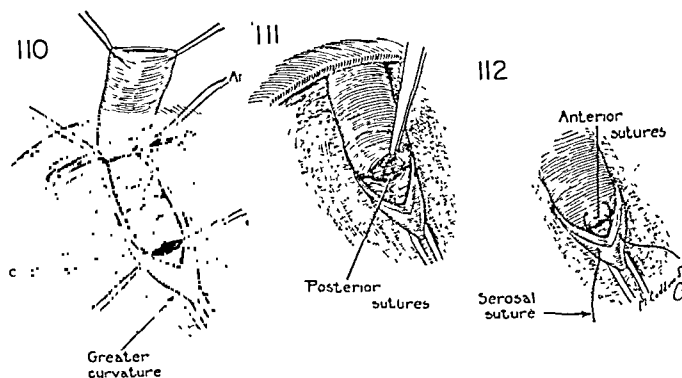
FIG. 110. An opening is made into the lumen of the stomach near the greater curvature. Traction sutures are placed in the opening of both the stomach and gall-bladder to facilitate the anastomosis.

FIG. 111. A direct anastomosis is made with interrupted sutures of silk between the fundus of the gall-bladder and the mucous membrane of the stomach.

FIG. 112. The fundus of the gall-bladder is anchored toward the greater curvature. The enterostomy clamp is removed.

bladder is not sufficiently elongated beyond the liver margin, an incision is made in the serosa, and it is detached for 3 or 4 cms. from the liver bed. The opening in the fundus of the gall-bladder is then increased to admit the tip of the adult finger. Traction sutures of fine silk are applied to either angle of the opening in preparation for the anastomosis. At this stage it is usually advisable to anchor the under surface of the gall-bladder as low as possible on the serosa near the lesser curvature because if this is delayed until the anastomosis is completed, exposure is very difficult. These sutures are usually taken deeper than those shown in Figure 109. After the lesser curvature is anchored to the under surface of the gall-bladder with interrupted silk sutures, an opening is made in the mucous membrane of the stomach near the greater curvature and its margins are held apart with traction sutures of fine silk. (Fig. 110.) A direct anastomosis is then made between the fundus of the gall-bladder and the mucous membrane of the stomach with one layer of interrupted silk sutures. Experimentally when the lumen of the gall-bladder was drawn within the lumen of the stomach, stenosis resulted. The same result followed in those patients on whom a similar technique was followed, making it evident that a direct anastomosis must be made to avoid stenosis.

(Fig. 111.) After the direct anastomosis is made and the patency of the lumen is verified by palpation the seromuscular flap is approximated in one layer with interrupted silk sutures which include a
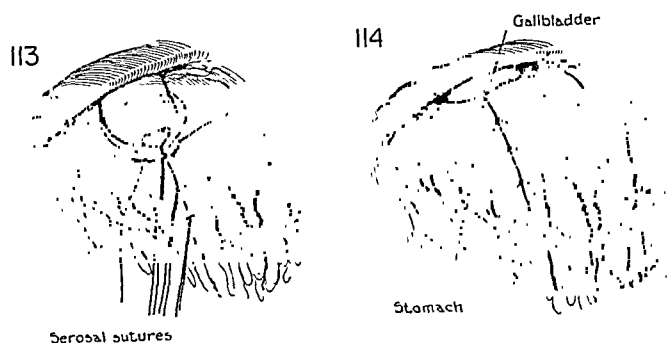
FIG. 113. The seromuscular flaps are approximated with interrupted sutures of silk which include a small bite of the gall-bladder wall.

FIG. 114. The gastric wall is closed toward the lesser curvature but not high enough to constrict the blood supply of the gall-bladder. It is not desirable to use a second layer of interrupted silk sutures in the seromuscular flaps.

small bite of the underlying gall-bladder wall. (Fig. 112.) These interrupted sutures are continued up toward the lesser curvature. (Fig. 113.) The flaps should not constrict the gall-bladder at the point of entry into the gastric wall near the lesser curvature. Undue constriction of the gall-bladder at this point is avoided to a large extent by making the cross incision sufficiently long. (Fig. 106.) Additional sutures may be taken between the gall-bladder and the anterior gastric wall if preferred. (Fig. 114.) The omentum is brought up to the site of anastomosis, and routine closure follows. As in any case of abdominal incision in the presence of malignancy and malnutrition, retention sutures in sufficient number should be included in the closure.

Because these patients are usually severely jaundiced the immediate postoperative concern is the control of the hemorrhagic tendency. Vitamin K therapy and transfusions are indicated. The diet should be limited for about one week to avoid undue tension on the suture line.

Although there was a hospital mortality of 25 per cent following some type of short circuiting operation in the presence of obstructive jaundice, the majority of the survivors experienced relief from pruritus, pain, and jaundice by the time they left the hospital and lived in relative comfort for an average of nine months.

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EDITORIAL

THE AMERICAN ASSOCIATION FOR THE SURGERY OF TRAUMA

FIRST PRESIDENTIAL ADDRESS

KELLOGG SPEED, M.D.

CHICAGO, ILLINOIS

WITHOUT attempting to go into the intimate, and soon to be historical, details of the organization of this association, which I hope my successor will do for us next year, it is my desire to set forth in an expositional manner what the principal aims, ambitions and uses of this organization may be and what its future intentions may develop. ·

In the rapidly expanding field of general surgery several schisms have occurred within the last thirty years, ending in a breaking off of certain specialties and narrowed fields, such as ear, nose and throat surgery, genitourinary surgery, neurologic surgery, thoracic surgery, with others—and even one body devoted quite entirely to the surgery of goiter: It is not the primary desire or intention of the American Association for the Surgery of Trauma to cause the formation of an additional and possibly narrowed-vision group of surgeons under a different label, but to attempt an amalgamation and calling back to the fold of the well trained general surgeon of those interested in the maintenance of high surgical skill and scientific development in the phases of surgery which have to do with trauma, its immediate and distant effects and complications.

As we know or shall learn from the history of our organization, practically all the members are simultaneously members of other

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American associations of different branches of surgery or the American Surgical Association itself. The press of time and the limitations of subjects at the annual meeting of many of these organizations has led to a great diminution in the number of papers concerning trauma submitted by title, and almost a complete absence, year after year, of papers of this character really read before the various meetings mentioned. This shoving into the background of trauma and its field in connection with all branches of surgery has not been because everything is known about the subject, but because vital and interesting physiologic studies and advances, coupled with anatomic study and daring have spread into every field of surgical research. This has led to so many reports and outbursts on physio- and chemicosurgical subjects in the literature, some controversial, some rapidly accepted and some patently unsound from their inception, that old fashioned trauma and its connections, forever with us like the poor, have jogged along, unheralded, poorly nourished and yet demanding a large share of hospital service, of operative time and skill in all parts of the world.

To correlate the newer advances and adjustments of surgical study and technique, the surgery of trauma now steps forth and demands recognition. The Association for the Surgery of Trauma has already attracted, and will continue to attract, the brightest surgically inclined minds. Its forum is opened to the problems of all phases of trauma and its consequences without developing any feeling of inferiority complex among essayists and discussers.

In this galaxy of surgical personnel before me, are men well informed in many of the problems of trauma in its connection with the various tissues and parts of the human body. Many of us apply the most advanced physiologic and pathologic information to what may appear a simple instance of trauma affecting our fellow-men. Consider for a moment the scope of this subject—its relation to shock and the mortality of accident, whether acquired by the impact from an automobile or the staggering blow from a surgical operation, possibly elective in character, and not primarily caused by injury. Consider the advance in study of the infections and gross lesions subsequent to trauma, the anatomic and physiologic studies of trauma of the human hand or skull or craniocerebral tissues. The magnitude of the field spreading out before this association becomes apparent and lends dignity to its birth.

These meetings should consequently be on parity with other American associations for surgical study and are intended to allow an outlet for information, practice, theory and research in connec-

tion with the surgery of trauma. They will induce all fellows of this association, as well as those influenced by them in teaching, in hospital contact and professional brotherhood to demand the highest grade of surgical art and practice for the enormous field of traumatic surgery. As I have stated, we aim to coalesce the best of surgical knowledge and practice and have no intention of adding any new board of surgical licensure, but expect each newly elected member to possess a certificate of ability obtained from the American Board of Surgery.

May I quote from the Presidential address of F. W. Rankin before the Southern Surgical Association in 1938: "The three major objectives which, I believe, the progress of modern surgery will demand in the future, are: First, elevation of standards of graduate instruction; second, adequate certification of surgeons; and third, the controlled hospital. Increasing specialization naturally evokes a number of questions: Do we have too many specialists: is specialization being gradually overdone? The answer to this, I think, must essentially be—no, we do not have too many qualified specialists— that is, if we designate as a specialist a member of the profession, who by training and experience will be accepted by his peers as expert in a particular field. There is no substitute for experience and only by prolonged apprenticeship in a given field may anyone become sufficiently qualified to be designated as a specialist."[1]

The American Board of Surgery has adopted a conservative arrangement of training years for the purpose of qualification before it—namely, five years of training after the interne year, three of which are devoted to intensive postgraduate study preceding eligibility for examination before the board. This qualification is useless without two compensating factors, first, recognition of the meaning and character of this qualification by the public, second, a proper and controlled enforcement of these standards of qualification by the hospitals. Appointments to the surgical staff of hospitals should be made solely from those thus qualified, or in the process of qualification, while under supervision.

The American Association for the Surgery of Trauma, most of whose members are diplomates of the American Board of Surgery, offers consequently a high standing forum for discussion of the problems of trauma in surgery and an opportunity to sift the merits of advances devised or reported. It must exert a wide spread influence on hospital standards, postgraduate teaching and oncoming surgeons.

[1] RANKIN, F. W. Presidential Address, Southern Surgical Association. *Ann. Surg.*, 107: 650 (May) 1938.

Its place in the sun of our chosen profession should be assured from the start. Our supreme effort must be properly directed in wise selections of programs, in regular attendance, in earnest contributions and in careful selection of members to succeed us.

It was Hippocrates who said:

"There is no necessity for much study, then, in order to set a broken arm, and in a word, any ordinary physician can perform it; but I am under the necessity of giving the longer directions on this subject, because I know physicians, who have the reputation of being skilled in giving the proper positions to the arm in binding it up, while in reality they are only showing their own ignorance. But many other things in our art are judged of in this manner, for people rather admire what is new, although they do not know whether it be proper or not, than what they are accustomed to, and know already to be proper; and what is strange, they prefer to what is obvious."[1]

[2] HIPPOCRATES. *Medical Classics,* 3: 171 *(Oct.)* 1938.

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SKIN AND FASCIA GRAFTING

ADDISON G. BRENIZER, M.D.

CHARLOTTE, NORTH CAROLINA

I AM dealing first, in brief, with the familiar subject of skin graft-
ing and then with the less familiar subject of fascia grafting
in some of its uses.

FIG. IA. Large ulcus cruris grafted with full thickness pinch grafts.
Two conditions are important in these cases: (1) tying off the
varicose veins, which can be done by ligating them, piercing
through the skin without incision; and (2) having fine, closely
knit granulations to graft upon.

The oldest and most universally used method of skin grafting is
that of Reverdin. Its chief advantages are that it is very easy,
requiring nothing more than a straight needle and a razor blade and
that the small pinches will remain on surfaces grossly infected. A
simple procedure, it can be used on debilitated patients. There are
objections, particularly for exposed parts. It gives a mottled or
mosaic appearance, is irregular in surface and height on account of
the lack of uniformity in cutting and the fact that the small graft
itself is cut thicker in the center than at the periphery. There is a

scar around each graft and the method is of little value in the correc-
tion of cicatricial contractures. (Fig. 1.)

The very objections to the Reverdin graft may be of advantage,
however, as in the following case (Fig. 2):

The patient received a spiral cut around his head, scalping him except
for two narrow pedicles. The scalp and skull were cleaned and replaced.

B   C   D

FIG. 1B, C, and D. A case of lymphangitis before and after a Rogers-Kondoleon operation
where long strips of skin were cut from the trochanter to the external malleolus and
from the crotch to the internal malleolus It is essential in these cases to treat the edema
of the legs as otherwise the grafts will finally slough away. The edema in leg ulcers is due
more to lymphangitis than to phlebitis. Many so-called varicose ulcers require, in
preparation for a graft, either a Rogers-Kondoleon or a modified (not so extensive)
Kondoleon

They healed over most of the skull, but on account of poor blood supply,
the whole area over the calvaria sloughed, leaving the top of the head bare,
down to dry bone. Small holes were drilled through the outer table of the
skull and granulations from the diploe allowed to grow up through these
holes. The granulating mass was curetted down as the outer table was
pushed up and could be removed in large plaques. As the granulations grew
and the top was skimmed away with a curette, the depression on top of the
head was being filled in, layer-cake-wise. Then Reverdin grafts were taken
rather deeply from the patient's very hairy chest. The roughness of the

graft and the stubble of hair put out by the grafts offered a very nice mesh over which he could brush his hair from the side. This held the hair much more securely than a toupee could be placed. I should be very proud to call this "Brenizer's operation for bald head"!

A   B   C
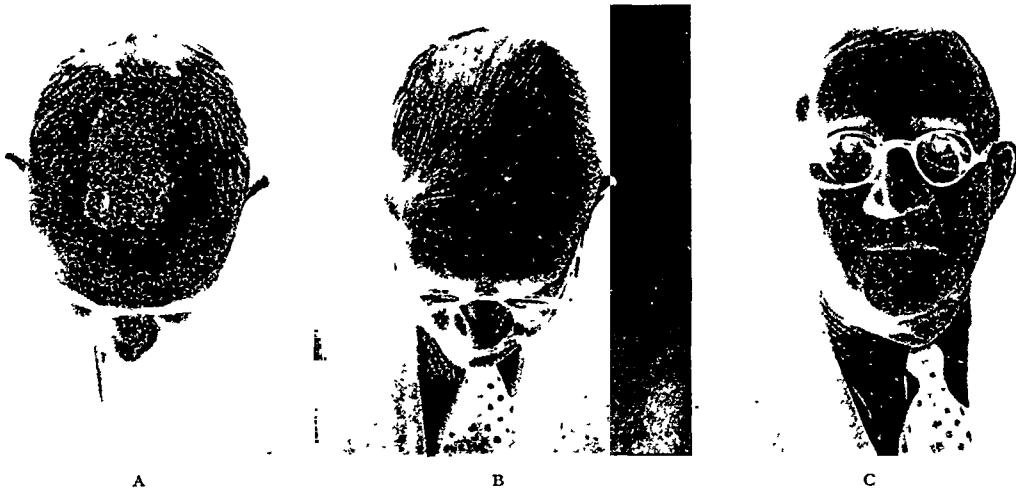
FIG. 2. A, denuded skull grafted with full thickness pinch grafts containing hair follicles. The granulating base is secured by boring small holes through the outer table of the skull, allowing the granulations to grow through the diploe, removing the outer table and repeatedly curetting the granulations until they are built up, layer-cake-wise, to the level of the skin. A small area of outer table has been left and must be removed for complete healing. B, hair brushed over the fine stubble of hair from the pinch grafts. C, front view.

The method described by Ollier in 1882 and Thiersch in 1886 has served us all admirably. Those of us who worked with Halsted were accustomed to cover the denuded area not embraced by the flaps in breast amputations. We found that not only did this area heal well, but the place from which they were taken also healed. However, the skin on the area was thin, the inequalities of the background of muscle and ribs showed through the grafts, and the grafted area contracted and wrinkled. Now, with breast flaps properly and extensively cut, we rarely leave an area not covered or produced by slough that cannot be covered with a few pinch grafts.

It is very difficult to find a perfect material in plastic surgery. However well the operation may be done, the patient invariably thinks that since you have done that well, perhaps if you had been a better surgeon or more careful with his precious flesh, you could have done better.

The full thickness graft described by Wolfe of Glasgow in 1875 was a decided improvement over anything yet offered. The best cosmetic results can be obtained with it and it is therefore used on the face. It stands up well under trauma and offers maximum

mobility over joint surfaces. Such grafts also contract less. Objections, however, there are: Even full thickness skin grafts, like all others, contract somewhat, and most meticulous care must be taken

A   B
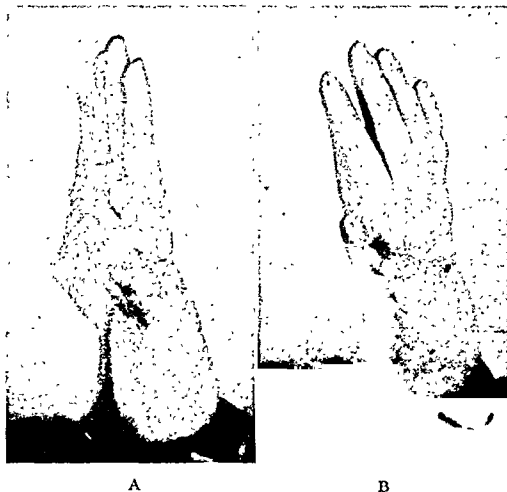
FIG. 3. A, result of severe automobile accident. Debrided, tendons united and grafted immediately with split skin grafts. B, dorsal view.

A   B

FIG. 4. A, twenty years after fascia and bone grafts from tibia had been inserted into a large skull defect, showing perfect result. B, profile.

for their protection against infection. Damage to blood supply and infection will account for many more losses than in the thinner grafts. Cutting the full thickness graft is tedious, the application is difficult because the proper pressure and tension must be attained

and maintained for a period of several weeks. A provision against loss of graft can be made by tunneling or tubing the full thickness graft for the development of better blood supply and the main-
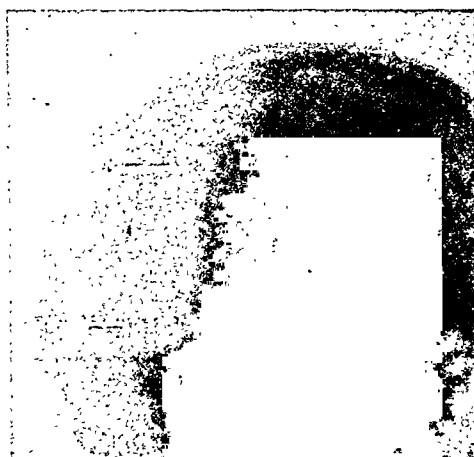
FIG. 4C. X-ray of same patient shown in Figure 4. The whole lighter area in the occipital region shows the three large bone shells from the upper tibiae. Indicates how large a defect was covered.

FIG. 5. A much smaller defect filled with bone from the tibia.

tenance of an arterial pedicle. However, there is frequently no choice of method to avoid these tunnels and tubes, though the area of skin is reduced by contracture and the skin in such a shape is more difficult to handle. In burn contractures around the neck restoration of an approximate configuration may be secured by several

FIG. 6. Shrunken and collapsed cheek resulting
from burn, filled in with fascia and fat. Allow-
ance is made for shrinkage of the fat and fascia,
but how much allowance to make is guesswork.

FIG. 7. Paralysis of facial nerve from
opening of parotid abscess. The face
was lifted with three strips of fascia,
run from an incision in the hairline
down to the external canthus, the
corner of the mouth and the border
of the mandible. A marked ectropion
of the lower lid was corrected by a
suspension strip of fascia from inter-
nal to external canthus.

FIG. 8. Child with cleft lip,
cheek, palate, jaw and
orbit. Soft parts reinforced
and held with fascia.
(From Brenizer, in Ann.
Surg., 107: 692, 1938)

operations of skin shifting, pedicle grafts, split skin and pinches. Under the chin and in the axilla devices must be used to hold the graft in place.

FIG. 9. A, harelip. By the use of fascial bands or ribbon catgut, placed between the mucosa and muscularis of the lips, tension is taken off the suture line and the lips may be united with muscle sutures of fine catgut and a subcuticular stitch of fine malleable wire. The soft parts may be placed and maintained in proper configuration with minimum scarring of the lips. The scarring of the mucous membrane readily repairs and is not visible. The slight increase in bulkiness of the lips soon subsides. B, harelip and cleft palate, ribbon catgut be'ng used for repair of both lip and palate. Union, configuration and function all excellent. Lip slightly bulky three weeks after operation. c, good union, configuration and function in harelip and cleft palate; no surface scarring. Deficient right ala and irregularity in vermilion border. (From Brenizer, in *Ann. Surg.*, 107: 692, 1938.)

Gillies[1] has done more to forward the tubular pedicled graft than any one else. Maltz[2] has modified the Gillies tubular graft by turning the raw side, rather than the skin side, out. Dean,[3] calling his graft a dermigraft, first cuts away a Thiersch graft and then, beneath this, a split skin graft or full thickness graft. It has already been mentioned that after a split skin graft has been taken and the skin has regenerated, another split skin graft can be taken from the same area.

Earl C. Padgett of Kansas City uses a calibrated skin graft and has an instrument for cutting skin of any thickness or pattern from anywhere in the body.

Blair[4] of St. Louis, on December 11, 1928, before the Southern Surgical Association, showed the split skin graft and suction retractor; since then, he and Brown have given many illustrations of its use. Brown's paper[5] before the Southern Surgical Association in 1937

was a superlative account of grafting split skin and full thickness skin on the hand.

Blair has pointed out that the split skin graft has more of the

A   B
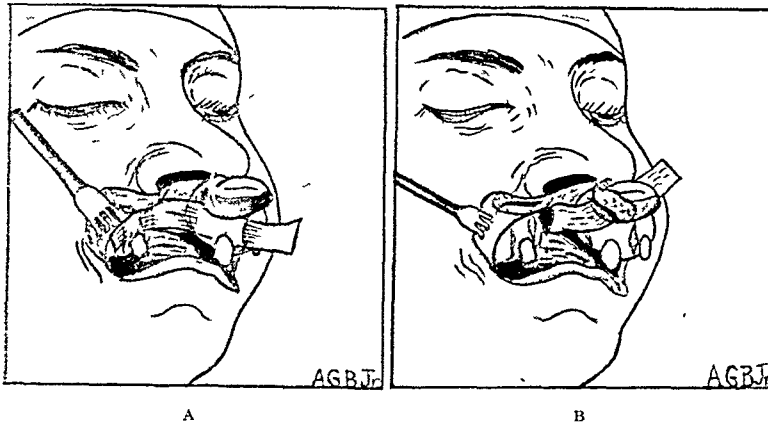
FIG. 10. A, vomer-premaxilla sectioned at the neck or not sectioned. Strip
of fascia inserted through opening in front under mucoperiosteum of pre-
maxilla and out and under mucoperiosteum of premaxilla and alveolar
process, where it is sutured. The fascial band is drawn upon tightly and
attached, in similar fashion to the opposite alveolar process. B, second
strip of fascia passed under philtrum between mucosa and muscularis and
stitched out into the lips on both sides between the mucosa and mus-
cularis, pulling lips together and relieving tension on the suture line.
(From Brenizer, in *Ann. Surg.*, 107: 692, 1938.)

A   B
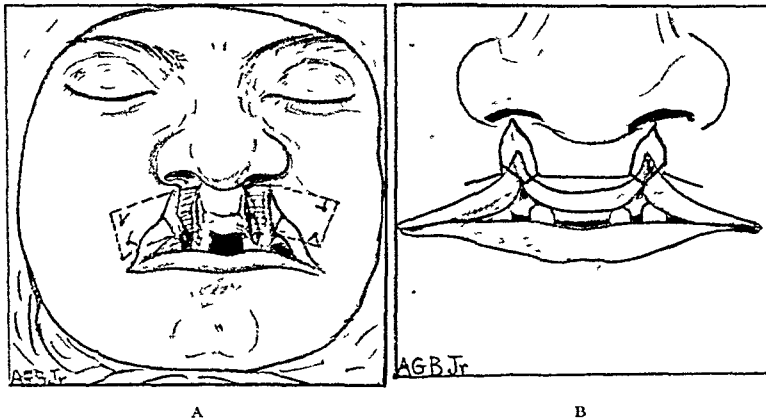
FIG. 11. A, first operation performed upon double harelip, using fascial
band. B, pattern of second operation where only the skin and muscularis
are incised and the mucosa is left intact. (From Brenizer, in *Ann. Surg.*,
107: 692, 1938.)

good than the bad points of either the Ollier-Thiersch or the full
thickness graft. With sufficient practice with Blair's suction retractor
and knife one can cut a graft of almost any thickness and from 25 to
50 square inches in area.

The donor area must be free from pustules (for infection accounts for 90 per cent of the failures) and non-hair bearing. It is cleaned with ether, metaphen, and alcohol, and is covered with a thin layer of

A   B
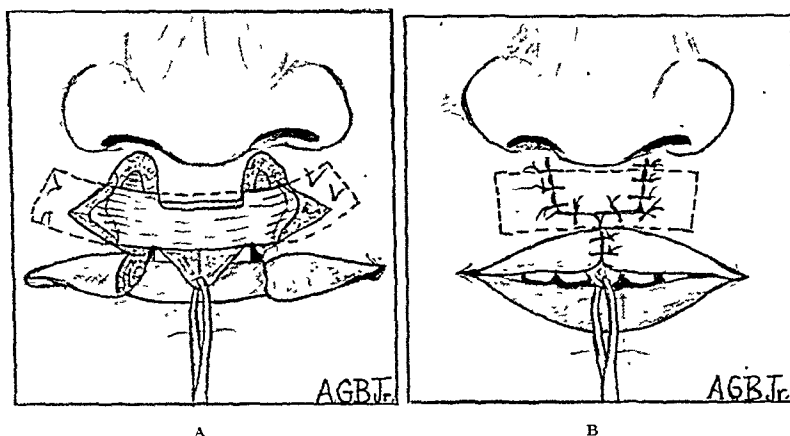
FIG. 12. A, band of fascia or ribbon catgut may or may not be used in second operation. The mucosa at the vermilion border is used to point up the lip. B, union of the pattern. When the band of fascia is employed, surface stitches may be avoided. Note the tip of mucosa crowning the vermilion border and pointing up the lip. (From Brenizer, in *Ann. Surg.*, 107: 692, 1938.)

vaseline so that the suction retractor will slide easily over the skin. As the skin is sucked up to a straight line and leveled against a countertraction, it is cut with a razor-sharp knife, making bold strokes out and back. One single graft, somewhat larger than the defect to be covered, is the goal desired.

The area to receive the graft must likewise be clean. It should be washed with soap and water, removing all scales and crusts, and is covered for a few days with warm saline solution. The granulations are curetted and cut away, down to the scar base, and the borders excised. All bleeding must be scrupulously stopped. If it is not completely stopped, a day's wait is in order. The graft should be sutured to the skin borders, loosely enough to avoid tension but taut enough to cover and fit the underlying structures. Rather than put stab wounds in the graft, it is advisable to insert small glass tubes between sutures at the skin border. The wound is dressed with several layers of xeroform gauze, a thicker saline gauze and a moist sea sponge cut to extend just to wound edge. The dressing is held with a pressure bandage; this pressure should be firm but not too hard. The dressing is changed about the sixth day, any serum evacuated, stitches removed, padding of gauze and sponge dispensed with and the wound covered with saline dressings for another week. Figure 3 is an

illustration of the ideal treatment of a fresh wound with good results. The back of the hand had been badly mangled in an automobile wreck and the distal phalangeal bones of the thumb were held

A   B

FIG. 13. A, double harelip markedly projecting and deviating premaxilla. Diagram of fascial bands, section of vomerpremaxilla at neck. B, final result (From Brenizer, in *Ann. Surg.*, 107: 692, 1938.)

by a flexor tendon. The hand was debrided, the tendons and even the nerves seen were united, and a free graft was done immediately. The result was excellent for function and fair for looks.

The area from which the skin is taken is covered with vaseline gauze and allowed to heal from the derma and hair follicles. When covered with epithelium this same area could be used repeatedly.

As far back as 1911 the experiments of Davis[6] are recorded, indicating most of the uses of free fascial flaps, except on body cavities and known infectious localities, such as the mouth and nose. In 1937,[12] I presented a report on the use of fascia in the repair of cleft palate and harelip, demonstrating two bony palates where fascia was used.

We have used fascia under bone grafts in skull defects,[7] to pad the cheeks, as bands to support the cheek and ectropic lower eyelid,[8] between mucous membrane and skin of the face and cheeks to support them, in harelip and cleft palate to support and relieve

tension on these structures and to have at least two bony palates,[8,12] to cover resected joint ends, and, in addition, as flaps and suture material in bone grafts[11] and in repairing the abdomen, particularly in herniae.
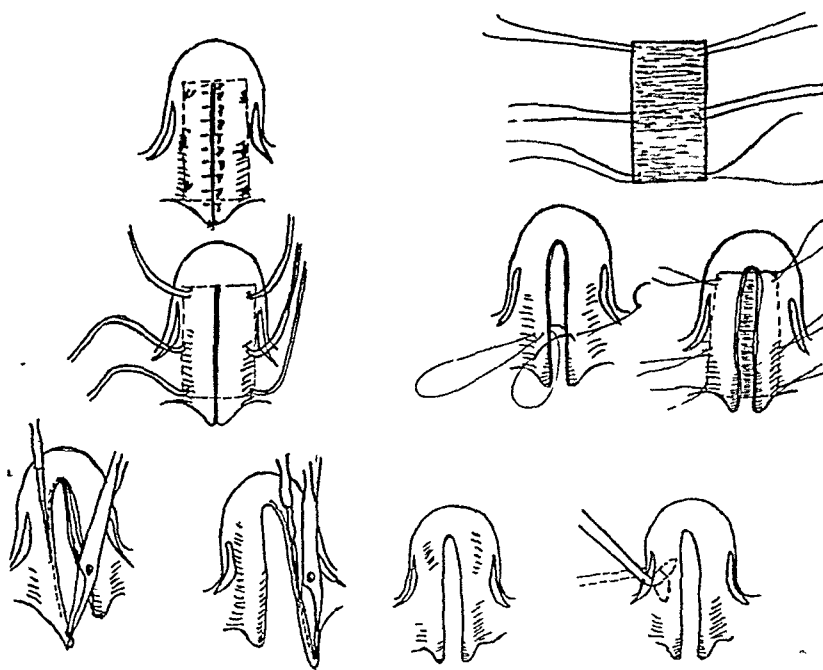
FIG. 14. The fascia first used in the cleft palate was a quadrangular strip placed in behind the mucoperiosteal flaps of the Langenbeck type of operation, or between the buccal and nasal mucoperiosteal flaps of the Victor Veau operation. (From Brenizer, in *Ann. Surg.*, 107: 692, 1938.)

In a paper[7] in 1916 I suggested that fascia be used to replace the dura. In a soldier, who was shot in the back of the head a few minutes before the Armistice was signed, the occiput and right parietal were badly broken and shattered. He lost some more bone and the scarred brain bulged through the opening. The brain was reduced by ventricular puncture and held back by a strip of fascia and then covered by three bone shells taken from the upper end of the tibia. The result was excellent. (Figs. 4 and 5.)

The boy shown in Figure 6 was badly burned over his right cheek, the structures being cooked and shrunken. He wore in his mouth a pad of cotton, which suggested to me the idea of making an incision along the lower border of the mandible and separating the skin from what was mostly scar and mucosa, thereafter filling the cheek with fascia and fat.

The girl in Figure 7, when a child, had an abscessed cervical gland opened by a doctor who, at the same time, cut the facial nerve. Her lower lid hung down in an ectropion; her whole face sagged. A

A   B

FIG. 15. A, first case, operated on at the age of three months, now 18 years old, retaining the fascial strip and showing a bony hard palate. Periosteal cells conveyed across the cleft in the hard palate. B, second case with bony palate, following fascial implant. (From Brenizer, in *Ann. Surg.*, 107: 692, 1938.)
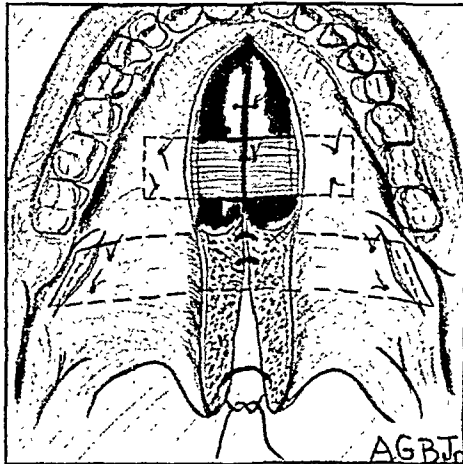
FIG. 16. Narrower strips of fascia or ribbon catgut, with a tie of fascia or ribbon catgut in the velum are now employed with good results. (From Brenizer, in *Ann. Surg.*, 107: 692, 1938.)

strip of fascia was passed between the skin and the musculature of the lower lid, suspending the two ends at the canthi. Three strips of

fascia were let down from an incision in the hairline, under the skin, to the ala, the mouth corner and lower border of mandible. This was a sort of a face-lifting scaffolding, which has remained fairly effective until this day.
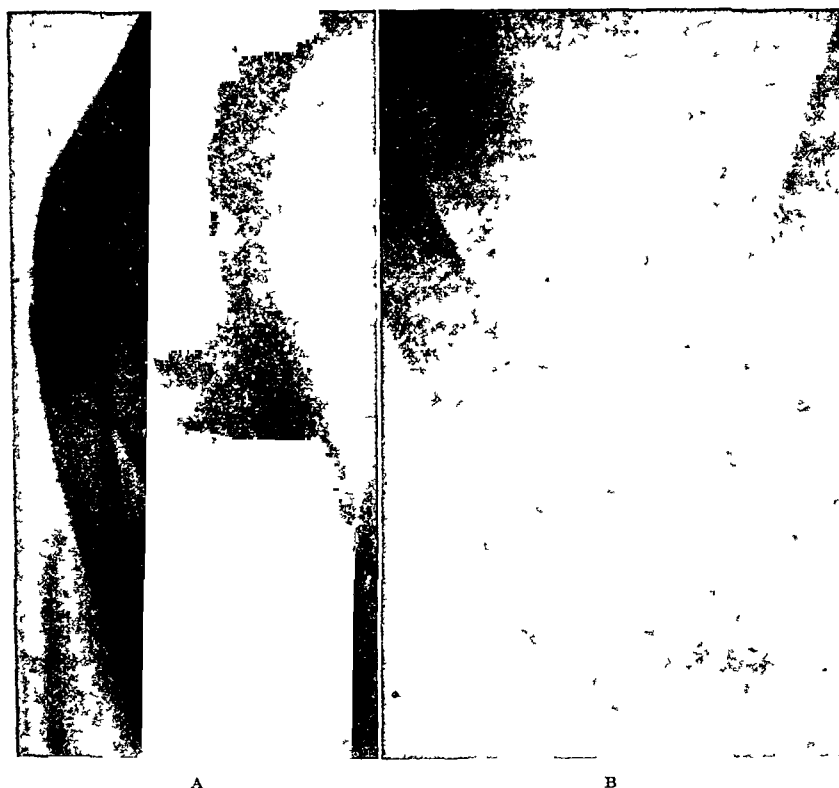
FIG. 17. A, resection of elbow with interposition of fascia. Elbow in extension. B, same case. Flexion of elbow. This same procedure has been used on hips, knees, etc.

A patient born with a cleft of the whole cheek, palate and maxilla up through the orbit (Fig. 8) had fascial bands placed between the mucosa and muscularis to relieve tension on the stitch line and reinforce the cheek. Following Dr. Blair's visit and sojourn with me during the World War, we used similar fascial bands to support the cheeks on my service at Base Hospital # 6, A.E.F.

Some of the cases shown in Figures 8 to 16 were shown before the Southern Surgical Association in December, 1937.[12] In these cases the fascia was inserted between the mucosa and muscularis in the lips and between the periosteum and bone in the palate and if Victor Veau's operation was done between the buccal and nasal flaps.

In four cases, comprising two ankylosed hips, an ankylosed elbow and a bunion, fascia was interposed between the resected and apposed bone ends. (Fig. 17.)

In a previous paper[11] I reported on the use of fascial ties instead of kangeroo tendon and chromic catgut.

A large pendulous panniculus adiposus with bulging abdominal

FIG. 18. Large watermelon slice taken from the skin and fat of the abdomen. Fascial planes plicated. Plication well aided by using strips of fascia taken from the planes. This case is a reminder of many uses of fascia for replacement, support, etc.

walls illustrates the type of case in which fascia is used in internal corsetry or in inguinal and incisional herniae. The panniculus shown in Figure 18 was so large that when the patient, a singer, reached a high note, an enormous load of skin and fat would suddenly shoot from under her corset. In these cases, before there is any tightening of the fascial planes, a large watermelon slice of skin and fat is removed. In one of these cases the operation was begun for me by a cow, who nicely horned up the whole panniculus, from the inguinal regions to the navel. A number of these patients have expressed their gratitude for the shrinking of the figure, if for nothing else.

CONCLUSION

The patient himself offers a handy and effective material in his skin and fascia, to relieve his own misery and embarrassment and to bring to the surgeon a remarkable mixture of abuse and praise.

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THE USE OF SMALL DEEP GRAFTS IN THE REPAIR OF SURFACE DEFECTS

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Introduction. The healing of lesions in any location due to the break in continuity of tissues, whatever may be the cause and however great or small the lesion may be, is the foundation on which all surgery is based.

The healing of denuded areas following destruction of surface tissue has always interested me a great deal, and in this connection I began to experiment nearly thirty years ago with a type of graft which I have called the "small deep graft." During this period, I have written several papers on the subject, and anyone interested in the story of the development of this graft and in the literature is referred to these articles.

Those of us who have to deal with large surface losses due to burns, industrial injuries or traffic accidents are often confronted with the question of how best to hasten the healing. In a comparatively few cases immediate grafting must be considered, but in the large majority of cases it is necessary, for various reasons, to delay the use of grafts until a later period when there is more likelihood of successful results.

In this communication, attention will again be called to the value of very small grafts in hastening wound healing, particularly those grafts which are slightly larger and contain more of the full thickness of the skin than the very thin small grafts advocated by Reverdin.

Reverdin first described his graft in 1869 as a pure epidermic graft, but later said that the title "epidermic grafts" was not perfectly correct, "as the transplanted bit is composed of the whole epidermis and a very little of the dermis." In other words, it was the thinnest graft which he could cut.

The small deep graft differs from the Reverdin graft in that, although the margins are quite thin, the thickness gradually increases so that at the center, the graft usually includes the full thickness of the corium. These grafts differ in thickness from true Reverdin grafts practically as much as whole thickness grafts differ from true Ollier-Thiersch grafts and should not be confused with thin Reverdin grafts.

At one time Reverdin grafts were obtained by pinching up a superficial bit of skin with forceps and pinching it off with scissors. On account of this pinching method, the grafts were called pinch

A   B
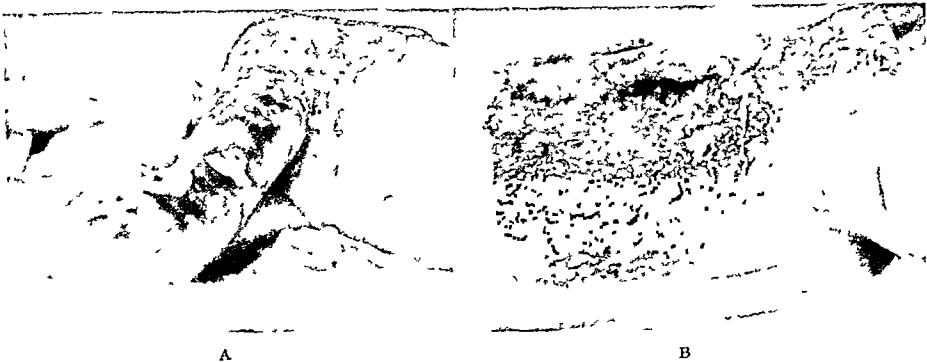
Fig. 1. Old unhealed burn in an 11 year old boy. Duration one year. A, note extent of involvement. Thigh flexed to more than right angle. Leg flexed on thigh. Fusion in groin. B, fusion of arm and forearm to chest wall, obliteration of axilla.

grafts. One frequently hears the term applied to small deep grafts, but this is incorrect, as small deep grafts cannot be properly cut by pinching off with forceps and scissors. Furthermore, Reverdin grafts, which are now seldom used, are no longer cut by this method on account of the unnecessary trauma to the tissues. It seems rational, therefore, to abandon the term pinch graft.

Nomenclature. The term epidermic graft is to be found in almost every article on skin grafting in which nomenclature or types of grafts are considered, but this term is inaccurate as there is actually no such thing as a true epidermic graft. There is no instrument as yet available which can cut pure epidermic grafts without some of the corium being included. As a matter of fact, a pure epidermic graft can be obtained in regions ordinarily used only by forming a blister, which raises the epidermis alone, and then by trimming this blister off and using it as a graft.

In a general way skin grafts may be grouped as thin and thick. In the thin group are those grafts which are made up of the epidermis and the tips of the papillae of the corium. True Reverdin grafts, 2 or 3 mm. in diameter, and true Ollier-Thiersch grafts (thin films of varying size) belong in this group; these are the thinnest grafts which can be cut. In the thick group are those grafts which are made up entirely or partly of the full thickness skin. Small deep grafts, whole thickness (Wolfe-Krause) grafts and sieve grafts belong to this group.

Other grafts very much used at the present time are obtained in the same way that Ollier-Thiersch grafts are cut, and in reality are

simply thick Ollier-Thiersch grafts. These are variously termed split grafts, intermediate grafts, razor grafts, mid-thickness grafts, etc., according to the taste of the author.
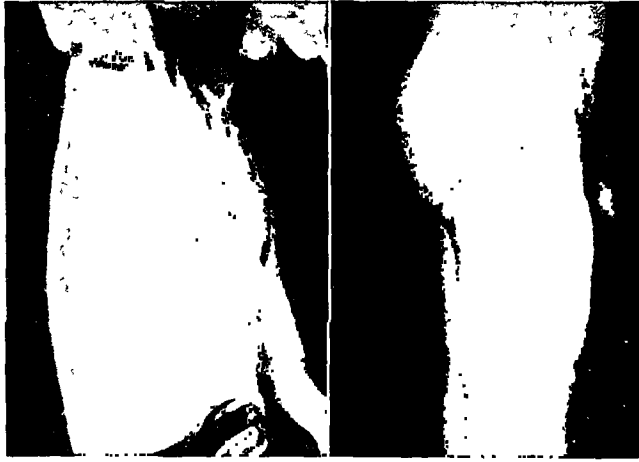
FIG. 2. Same case as in Figure 1 after nine and one-half years. The contractions were relieved by incisions and traction. The wounds were healed with small deep grafts. Note the condition of the chest wall and axilla. Slight recontraction in anterior axillary line. The leg and thigh can be fully extended and there is no evidence of recontraction. The size of these areas and the precarious condition of the patient precluded the use of any other type of graft. The appearance of the grafted areas compares favorably with that resulting from the larger types of graft.

Source of Small Deep Grafts. Auto or self grafts are almost uniformly successful when placed on a granulating surface which is in proper condition.

Iso, or homo, or same species grafts are occasionally permanently successful although it has been said that this is a biological impossibility. They will nearly always take on a suitable surface and will live and spread and stimulate growth from the edges, but usually in about three weeks, following a more or less severe anaphylactic reaction, they will melt away overnight. At one time, we used iso grafts quite frequently, but in recent years they have been rarely employed.

Zoo, or hetero, or animal grafts will frequently take at first, but are seldom, if ever, permanent.

Small deep grafts may be cut from any available portion of the skin, but where possible they should be taken from areas which are ordinarily covered by the clothing as the multiple small scars which are left after healing are more or less unsightly. We can almost

always find a usable area of normal skin, however extensive the destruction may be. The region from which the graft is taken has little if any effect on the result, but my preference is for the upper

Fig. 3. Old unhealed burn in a 7½ year old girl. Duration thirteen months. Appearance of the granulating wound of thigh and knee. Note flexion due to scar contracture at knee and hip.

anterior surface of the thigh, if available, as the skin is not too thick and has about the right tension.

Surface on Which Grafts May Be Placed. Small deep grafts may be placed on fresh wounds; on surfaces from which the granulations have been entirely removed; and on surfaces from which the granulations have been partially removed by slicing off the undesired portions. In most instances, however, they are placed on undisturbed clean, flat, firm healthy granulations, as there is no pain in the process; no danger of stirring up infection; no loss of blood, which is an important point in patients already much depleted.

Preparing a Granulating Area for Grafting. Each surgeon has his own ideas as to when granulations are ready and how to prepare them. Suffice it to say that when the granulations are clean, firm, flat and rose pink in color, and the bacterial count is low or negative, the wound is ready to receive the grafts.

The question is frequently asked, "How do you get a granulating wound into this condition?" The procedure depends on whether the granulations are boggy and exuberant or whether they are scanty and sluggish. Some of the methods which have proved useful to me are as follows: When the granulations are boggy and exuberant, free application of silver nitrate, either in stick or as a saturated solution is often effective. Sulfate of copper in the crystal form or as a saturated solution is also useful. Slicing the granulations off with a scalpel or trimming them closely with curved scissors, followed by the application of silver nitrate or copper sulfate, is quicker and more positive. Thorough curetting of the granulations may in certain

instances be the method of choice. Continuous compresses of bismuth, or iodoform gauze, or plain gauze saturated in sterile glycerin may be helpful.
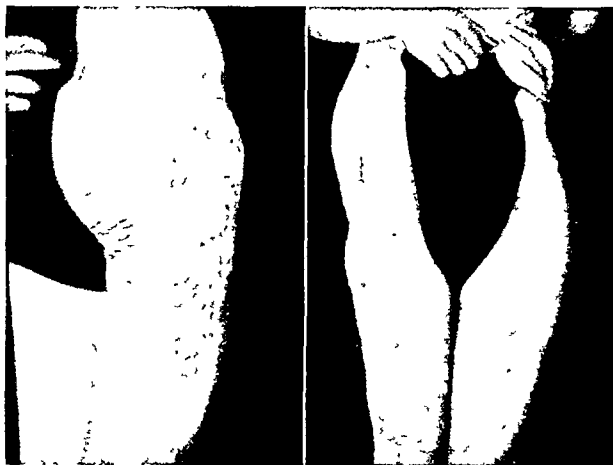
FIG. 4. Same case as in Figure 3 after twenty-one years. There is strong, flexible healing with no tendency to re-contracture. There has been no interference with the growth of the injured leg, as compared with the normal one. The grafted skin is freely movable over the under-lying tissues. The appearance is good. The patient says that she has no discomfort whatever, that function is normal, and that she does not realize that anything was ever wrong with her leg and thigh.

Edematous and exuberant granulations may be flattened by evenly applied pressure. A sheet of rubber or rubber protective with small darts cut in it is placed over the granulations. Then sterile sea sponges or rubber sponges are applied and snug pressure is induced with bandages, elastoplast or adhesive plaster. Sometimes pressure produced by tightly applied adhesive strips or by 4 inch elastoplast bandages is sufficient to flatten the granulations.

Although the proper use of Dakin's solution by the Carrel method is rapidly being lost sight of, I still use this method with a great deal of satisfaction, and granulations which are exuberant and edematous will often in a few days shrink and become firm and healthy. Dakin's solution, used by the haphazard methods fre-quently seen today, is usually ineffective. Exposure for a short time for several days in succession to ultraviolet light may aid in the sterilization of granulating areas, and will also help in flattening out exuberant granulations.

When the granulations are sluggish, stimulating treatment is necessary. Painting the wound with 2 per cent nitrate of silver is

sometimes helpful. Compresses saturated with normal salt or boracic acid solution may be effective. Compresses saturated with sodium chloride solution 15 to 20 per cent or with glucose solution 20 to 40

A   B
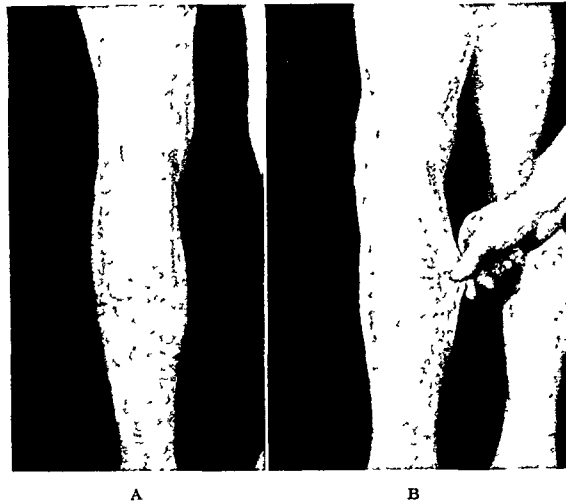
FIG. 5A AND B. Male, 42 years old, twenty-three years after grafting of an extensive deep burn of the leg and thigh with small deep grafts. The grafted area is soft and movable, and as stable as normal skin. There is no tendency to scar contracture although the flexor surface of the thigh, knee joint and leg was grafted. The contour of the popliteal space is normal in appearance. The healed area is loose and can be easily pinched up. The appearance is about as good as that which would have followed the grafting of such an extensive area with the larger types of graft. The small scars of the wounds from which the grafts were taken can be seen on the other leg and thigh.

FIG. 5C. Same case as in Figure 5A and B.

per cent will often start granulations on a sluggish base. Gridiron incisions through the scar tissue base may stimulate the growth of

granulations. If the scar base is very thick and the wound old and sluggish, it is sometimes necessary to excise the entire base down to normal tissue with as much of the margin as is necessary. The application of plain or iodoform gauze saturated with either undiluted balsam of Peru or in combination with castor oil 1:3 is frequently helpful. Immersion in a continuous tub is often useful in cleaning and stimulating granulations. Heliotherapy in the form of electric light baths and real or artificial sunlight in suitable amounts are valuable in treating all types of granulations and will often stimulate growth.

Granulations are sometimes stimulated and cleaned up by painting the entire wound with saturated solution of silver nitrate followed immediately by full strength tincture of iodine which forms silver iodide. Painting the granulations with gentian violet 5 per cent in water, or with dahlia 2 per cent in water, is useful in certain cases.

Preparation of the Area from Which the Grafts Are to Be Cut. After the area is shaved, a thorough scrubbing with green soap and water followed by ether and alcohol is the most satisfactory method· of preparation for this area.

Preparation of Healthy Granulations to Receive Grafts. On the day preceeding the grafting, the wound should be carefully cleansed, and then dressed with a thick pad of plain gauze or iodoform gauze saturated with normal salt solution. This gauze pad is allowed to dry out, but immediately before operation, is thoroughly soaked with normal salt solution and is removed, care being taken not to cause bleeding. The skin around the granulating area is prepared by any method which has the surgeon's confidence. The wound is washed with ether followed by normal salt solution. A pad of dry gauze is placed over it and is pressed down firmly on the granulations. The dry gauze is peeled back from the granulating wound just before the grafts are applied, as the surface to be grafted should be dry, but not glazed, as is the case when the surface is left exposed to the air.

Anesthesia. General anesthesia is rarely used for cutting small deep grafts except in very nervous children or excitable adults. Ordinarily, they are cut under local anesthesia induced by nerve block or by infiltration, with ½ per cent novocaine containing four drops of adrenalin to the ounce. Moderate infiltration seems to have little if any detrimental effect on the healing of the grafts, but all things being equal, nerve blocking is preferable.

Operative Technique. Place the patient in a comfortable position on a bed or a well padded table. Approximate the area of skin required to supply the necessary grafts, and outline this area in the

shape of a square or rectangle with brilliant green 5 per cent in alcohol. This localizes roughly the area to be infiltrated. If one anticipates the excision of the area from which the small deep grafts are taken, in order to make a line scar instead of multiple small scars, then the outlined area should be in the form of an elongated ellipse, and the grafts taken from inside this outline.

After the desired anesthesia is induced, a bit of epidermis is picked up on the point of a shortened straight intestinal needle held in an artery clamp and is raised so that a little cone is formed. Sharp knives are essential for accurate graft cutting. By cutting through the tip of the cone with blade level we can obtain a Reverdin graft. When the base of the cone is cut through, the blade being slightly tilted downward until the center of the graft is reached and then turned upward and outward again, a small deep graft is obtained.

The grafts are preferably round or oval, between 4 and 5 mm. in diameter. They should not be larger. They are thickest in the center where the full depth of the skin is usually included and taper off toward the edges where they are quite thin. The subcutaneous fat can be seen shining through the central portion of the little wounds from which the grafts are taken. A thin rim of undisturbed epithelium should be left between the pits made in cutting the grafts.

The grafts when cut are dislodged from the needle on to a folded towel which has been wrung out in normal salt solution. This will remove any blood which may be on the raw surface. The assistant applying the grafts picks up each one on another needle, and, after peeling back the dry gauze pad from the granulations, places the grafts about 5 mm. from the wound edges and from each other, with the epithelial surface up.

After the grafts are in place it is essential that the thin edges be uncurled and spread out on the granulations and that in addition the thick portion of the graft be in close contact with the granulations at every point. This uncurling and close contacting is accomplished by firmly pressing directly downward on the grafts with a gauze pledget. After the grafts have been uncurled and pressed down on the granulations, any one of several methods of immobilization may be used. In one, which I prefer, the grafted area is covered with overlapping strips of rubber protective about 2.5 cm. wide in which v-shaped slits have been cut. These strips are long enough to extend out on the surrounding skin to which they are secured by a few drops of chloroform. Another method is to place over the grafted area strips of paraffined mesh secured to the skin by adhesive plaster, and over the mesh a single layer of xeroform gauze, 3 per cent.

Immobilizing can also be accomplished by stretching a single layer of wide mesh plain gauze tightly over the grafted area and securing the margins to the skin with adhesive plaster or collodion. Over the gauze a single layer of xeroform gauze is placed.

Further immobilization is obtained by placing over the protective or paraffined mesh or plain stretched gauze, a thin piece of sea sponge, cut to cover the grafted area. Over this a thicker and larger sea sponge is placed, which projects beyond the grafted surface, the whole being secured under even pressure by means of adhesive plaster and a bandage.

Another method which is extensively used after the grafts are in place, is to expose them to the air from six to twelve hours in order to set them. This is done either with or without immobilization by means of paraffined mesh or plain gauze. It should be noted that the same effect can be obtained by exposing the grafts to a hot air douche for a few minutes. I prefer the closed method as it is easier on the patient and gives the best immediate and ultimate results.

Dressing of the Donor Area.   Silver foil is the most satisfactory dressing for the area from which small deep grafts have been cut. Several layers of foil are applied and over this the porous paper which separates the leaves. Then a single thickness of gauze impregnated with 3 per cent xeroform ointment, and finally a flat gauze dressing secured with adhesive plaster and a bandage. This dressing is left undisturbed for about two weeks, and when it is removed the little wounds will usually be healed.

Postoperative Treatment.   Immobilization of the part and confinement to bed for a longer or shorter period, depending on the size and situation of the grafted area, are essential when dealing with large wounds. In smaller wounds in suitable situations ambulatory treatment may be used. When the grafts are placed on a granulating surface the first dressing should be done after forty-eight hours; when on a fresh wound, the dressing may be delayed for a week or longer. Frequently, the grafts placed on a granulating surface and dressed with rubber protective strips will be found bathed in a creamy secretion. The protective should be removed, and this secretion should be gently mopped up or irrigated with normal salt solution. Then protective strips and sea sponge are again applied and secured with even pressure. If paraffin mesh is used, it is left in place and removed at the second dressing. Within forty-eight hours after grafting, those grafts which will live become dusky pink with a deep blotchy area in the center. A narrow halo of newly formed epithelium can often be seen around each graft. Those grafts which do not take

are pearly white in color and will come away with the dressings in the course of a few days.

It is unnecessary to reapply the sea sponge pressure after the first dressing, although a snug dressing is desirable. When the grafts are growing satisfactorily and spreading, some bland ointment on old linen is useful. When the growth of the epithelium from the wound and graft margins seems sluggish, 8 per cent scarlet red ointment will often cause stimulation of the growth. Compresses saturated with Dakin's solution may be used to flatten them without harm to the grafts after the fifth day. As soon as the newly formed epithelium from the grafts and the wound edges has fused, stearate of zinc powder and exposure to the air are helpful. The grafted area should be protected from injury for several weeks. About three weeks after healing has taken place, gentle massage should be started and should be continued until the grafted area slides easily over the underlying tissues. Desquamation of the healed grafted area usually occurs and continues for several months. This can be controlled by the application of cold cream or some other bland ointment.

On an extensive wound, it is seldom that we find all the granulations in the same condition at the same time.   If a small portion of the wound is ready to receive grafts, then the grafts should be placed about 5 mm. apart over this area. If a large area is ready for grafting, small deep grafts may be placed about 5 mm. apart over the whole area or may be scattered over it, and then in a few days more grafts may be put in the spaces between. This is continued until the grafts are approximately 5 mm. apart over the whole wound. Other portions of the wound should be grafted as they come into suitable condition, always aiming to have the grafts in the end not much more than about 5 mm. apart. The reason for this is that when small deep grafts are placed at greater intervals than 5 mm. apart, the granulations between them may become exuberant before the epithelium from the margins of the grafts can fuse and cover them, and thus control the growth. Grafts may in this way be overwhelmed by granulations and in some instances are completely buried and eventually absorbed.

From each small deep graft, 4 or 5 mm. in diameter, epithelium will spread over an area from 2 to 2.5 cm. in diameter if the grafts are placed with considerable intervals between them. Although good results are often obtained under these conditions, the healing will be more stable if they are planted with spaces of about 5 mm. There is usually stimulation of epithelium from the wound margins and from

the margins of the grafts previously applied following each successive application of these grafts.

There is no limit to the number of small deep grafts which can be cut at a sitting. Naturally, it is a much more irksome procedure to the operator to cut and apply several hundred of these grafts at one time than it is to cut and apply a large Ollier-Thiersch or whole thickness graft.

Preservation of Grafts. The idea is prevalent that skin grafts of all kinds must be applied either immediately or within a comparatively short time after cutting. Years ago I became interested in the preservation of skin grafts and have experimented with the refrigeration of various types. I soon became convinced that there was no hurry in applying skin grafts and that they could be preserved quite simply. If the grafts were to be used within twenty-four hours, wrapping them in sterile dressings moistened with normal salt solution to keep them from drying out, and storing in the icebox was sufficient. If longer periods were desired, the grafts were stored in jars of sterile yellow vaseline or sterile albolene in the coil compartment of a mechanical refrigerator.

For practical purposes, iso or zoo grafts can be eliminated from the problem of refrigeration as in the majority of cases, even if immediately transplanted, they do not cause permanent healing. Ordinarily, if grafts are cut in excess of the number immediately required, they are preserved and can be used later. Sometimes more grafts than are needed are deliberately cut and preserved. Now and then, an operative wound prepared for grafting will continue oozing in spite of all efforts to check it, to such an extent that grafts which have been cut cannot be applied immediately with any assurance of success. In these cases, the preservation of the grafts until bleeding ceases is important and when the surface is dry, either later in the day or on the day following, the grafts can be applied.

In large granulating wounds, it is seldom that the entire surface is ready for grafting at the same time. In these instances, the area which is ready is grafted, and surplus grafts cut at the same time are refrigerated and can be used at any time another portion of the surface becomes ready to receive them without any more discomfort to the patient than that of an ordinary dressing. As a matter of fact, it is seldom necessary to preserve grafts for actual use for longer than three or four weeks, as after that lapse of time the patient is usually healed or is in suitable condition to permit the cutting of fresh grafts.

The grafts, when removed from the vaseline or albolene, are as pink as when they are put in and look quite fresh. They are either

wiped gently with sterile gauze to remove the vaseline or are washed with ether before being placed on the granulations. It was noted that while the refrigerated grafts would take, and the blood supply become established, that it was fully a week and sometimes longer after transplantation before any new growth of epithelium was observed from the graft margins, and that this growth seemed slower than from immediate grafts of similar type. Microscopically, no definite change was observed in the cells of grafts refrigerated as above described, even after several months. Immediate autografts are always to be preferred, but I also feel that refrigerated autografts have a distinct field of usefulness, and that excess material should be preserved in suitable cases and used as required. Refrigerated small deep grafts are permanently successful, and we have successfully transplanted them after five weeks.

Frequently, the treatment of large surface losses of tissue is turned over to inexperienced interns, as such surgery is considered unworthy of the personal attention of the upper house staff. As a matter of fact, however, the successful treatment of large surface lesions is in many instances a very much more difficult bit of surgery, and harder to handle than many of the so-called major surgical procedures. In certain hospitals the first operation assigned to interns is that of treating burned areas with small deep grafts, or a reasonable facsimile.

The grafts are in many instances improperly cut as to depth and size and distance apart; are cut from areas such as the front and back of the leg, arms, breasts, over the sternum, and from other exposed positions; are placed on areas where other types of grafts should be used; are frequently put on granulating areas which are not properly prepared to receive them; and are improperly spaced, dressed and treated. The procedure is simple, but frequently I have noted that the surgeons who give the instructions to the interns have themselves a very indefinite idea of what a small deep graft is and of the essential details necessary for success. In consequence, the grafting is done quite inefficiently, and the results are in proportion.

We frequently see grafts cut which are from 1 to 1½ cm. or more in diameter and which are erroneously called small deep grafts. These grafts are unsatisfactory for several reasons: The result is more unsightly; not so much epithelial proliferation is possible from the margin of a single larger graft as from several smaller grafts whose total area is the same; where available skin is scanty, more is wasted than when small deep grafts of the proper size are cut and properly spaced; and the resulting scars are more objectionable.

In surgery, as in every other type of work, there is a great deal of difference in the way two men will carry out the same procedure.

Poor results, both immediate and subsequent, are often due to the lack of care in the preparation of the surface to be grafted. One has only to look about in any hospital where small deep grafts are used, to see that this is true, as some men, frequently have poor results, while others, who are meticulous in their pregrafting preparation, are almost uniformly successful.

Often, however, we are forced by circumstances to place small deep grafts on granulations which are in unsuitable condition for grafting, and it is surprising how frequently they will take on these surfaces where no other type of graft could survive, except possibly the thin Reverdin graft. Care should be taken to avoid the transfer of infection from the granulating wound to the area from which the grafts are cut.

It should be an unbreakable rule that when any type of graft is contemplated, the area from which the graft is cut should be selected with the idea of conservation of graft resources for subsequent use, if it should ever be necessary. Of course, other grafts cannot be taken from exactly the same area from which small deep grafts have been cut, but this is the case when a graft of any type has been previously taken. It is regrettable that in many instances, large areas of skin are completely eliminated for future graft supply by the thoughtless or unskilled operator, using, for instance, the whole anterior surface of a thigh to supply fifteen or twenty small grafts. This cannot be charged against the method, but is due to the stupidity of the operator.

As a matter of fact, one advantage in using the small deep graft, besides starting islands of skin over a large denuded area, is that grafts are taken from a comparatively small skin area, and if they are properly cut with proper intervals between, a greater raw area can be healed from a smaller skin source than by any other method. This may be verified by giving actual measurements in several recent cases. An area 7½× 6 cm. yielded 108 small deep grafts; another 6½ × 5 cm. yielded ninety-one grafts; another 6 × 6 cm. yielded 100 small deep grafts. Now these grafts, which should be 4 to 5 mm. in diameter, may be scattered widely over a large granulating area. When, however, we take the 6 × 6 cm. area from which 100 grafts were obtained and place these grafts about 5 mm. apart on a granulating surface, which is as close together as grafts of this type should be placed, they will cause stable healing on an area a little over 10 cm. square. On the other hand, if we cover an

area 10 cm. square with a thin or thick Ollier-Thiersch graft, it would require the denudation of a normal skin area larger than 10 cm. square. If this 10 × 10 cm. area was to be covered with a whole thickness graft, it would require the excision of a graft of the same size, 10 cm. × 10 cm., to accomplish it.

An area 10 cm. × 10 cm. is comparatively small when we consider the large surfaces which are often destroyed in extensive burns, industrial and accidental injuries, and operative excisions. The denudation of an additional area of 10 cm. square, or even considerably larger, in obtaining a thick or thin Ollier-Thiersch graft or whole thickness graft is of little importance, but when we have areas many times 10 cm. square to cover with these grafts, the situation changes, as the larger the wound surface to be covered, the larger the area must be from which these grafts are taken, and in consequence, the greater the additional denudation of skin surface and the greater the operative procedure. The denudation of this extra amount of skin surface, in addition to the original large wound, is often a serious added burden to the already depleted patient.

Some surgeons use Ollier-Thiersch grafts, either thick or thin, for almost every wound requiring grafting, while others use whole thickness grafts, and still others use small deep grafts. The most rational procedure is to use the type necessary for the needs of each individual case and supplement with any other type desired. For instance, when we have a large granulating surface, it is frequently impossible on account of the condition of the patient or the lack of available skin surface, to cover it completely with either thin or thick Ollier-Thiersch grafts or whole thickness grafts, even if these types were to be preferred. In such a case, small deep grafts offer the solution of the problem as they may be applied here and there over the surface to start islands of skin, then in a few days other grafts are placed between those already applied and so on until the new epithelium covers the area.

The results as far as appearance is concerned are less satisfactory than those of successful large, thin or thick Ollier-Thiersch grafts or whole thickness grafts, which completely cover the defect. The healed area is spotted with small grafts whose centers are the full thickness of the skin. However, in the majority of instances, small deep grafts are used to induce rapid healing in areas which are ordinarily covered by clothing, and appearance is not particularly important. Where the proper technique is carried out, the permanence and durability of the healing is much more stable than

following thin grafts of any type, and as stable as the results of whole thickness grafts.

On account of the dotted appearance of a surface successfully grafted with small deep grafts, it is inadvisable to use them on the face and other exposed positions except to hasten healing in special instances where subsequent removal is contemplated. Sometimes a brownish pigmentation may form in the grafted area, but this also occurs when other types of grafts are used.

It must be borne in mind, however, that the appearance of a wound healed with strips or patches of whole thickness grafts or thin or thick Ollier-Thiersch grafts is also blotchy and is often more unsightly than a wound healed by small deep grafts. There is somewhat more shrinkage in the size of the original wound after healing with small deep grafts than after successful thick Ollier-Thiersch and whole thickness grafts.

The difference in the stability and character of the healing obtained after grafting with thin Reverdin grafts and with small deep grafts is almost as marked as that which is found between the results of thin Ollier-Thiersch grafts and whole thickness grafts. Wherever a small deep graft has taken, there is a permanent patch of the full thickness of the skin with all of its components, and these little patches of thick normal skin give stability to the healing.

It has been suggested that the healing of the little wounds left by cutting the grafts may be expedited by making oval grafts and suturing each wound. This is hardly necessary as the healing is usually prompt and the appearance of the scars of a sutured group of oval pits is little better than when suturing is not done.

The excision of the entire area from which small deep grafts are cut, with closure of the defect in a line, was advocated many years ago and abandoned, but has recently been recommended again. This necessitates a much more extensive surgical operation, and is really the same procedure as removing a whole thickness graft, which on a depleted patient is inadvisable. However, if the area from which small deep grafts are taken is to be excised, then it is unnecessary to leave a rim of epidermis between the pits made in cutting the grafts, and the grafts can be cut closer together.

Sometimes the scars from which small deep grafts are obtained thicken and become keloidal in character. This is an undesirable complication, and we do not know when it will occur or how to avoid it. Keloid also may develop in the scars left by the removal of thin or thick Ollier-Thiersch grafts and also in the sutured wounds from which whole thickness grafts are obtained. In other words, keloidal

scar tissue may develop in any scar from which any type of graft is obtained.

Occasionally in patients with a keloidal tendency, there may be a thickening of the scar between the grafts even if they have been placed quite close together. However, this thickening is more frequently seen when the grafts have been placed with intervals greater than 5 mm.

If small deep grafts are placed closer than 5 mm. apart, there is unnecessary waste of skin. The healed wound then has a cobblestone appearance, as the grafts do not tend to flatten out.

The objection has been raised that scar contractions may occur in areas healed with small deep grafts, but these can ordinarily be relaxed in due time without difficulty, when the scar has matured, and the patient is in proper condition. As a matter of fact, there is no type of free skin graft in which, after healing has taken place, scar contracture does not sometimes occur, either in the grafted area itself, or along the margins. This contraction is especially liable to occur on the neck, in the axilla or groin and around joints.

SUMMARY

It has been said that the results obtained by the use of small deep grafts are never satisfactory; that small deep grafts, often incorrectly called pinch grafts, should never be the method of choice; that by the use of these grafts the donor site is made useless as the source of other grafts, and that the method is a surgical error.

My experience has been that if small deep grafts are properly cut and are *really* small deep grafts; if they are used in the proper situation on a surface which is suitable for them; if they are properly spaced; if they are properly dressed; if the after-care is as it should be—the healing will be stable, strong and permanent, and there is no type of graft which gives more uniformly satisfactory results. Furthermore, small deep grafts will in many instances take on surfaces on which no larger graft could take.

If the best results are to be obtained with small deep grafts, several points must be observed. The granulating surface on which the grafts are placed should be healthy, clean, flat, firm and rose pink in color. The grafts should be cut without unnecessary trauma, should include the full thickness of the skin at its center and should be no larger than 5 mm. in diameter. In cutting, a narrow rim of undisturbed epidermis should be left between the little pits, and in this way a large number of grafts can be obtained from a very small area of skin. In fact, a greater raw area can be healed from a smaller

skin source than by any other method. The grafts should preferably be placed on the granulations with a space of about 5 mm. between them, although frequently excellent results are obtained when the spaces are greater. They should be pressed down firmly so that the thin edges will uncurl and so that every portion of the graft will be in close contact with the granulating surface. The grafts should be immobilized until the new blood supply is assured, and this is best accomplished by the closed method of dressing.

My own experience and that of many other surgeons during nearly thirty years has convinced me that the method is that of choice in a great many instances, and I feel that those who have not had good results, either do not know how to use this type of graft or do not take the trouble to use it properly.

In my own work I use small deep grafts constantly, with the greatest satisfaction, and find the method indispensable when dealing with large losses of surface tissue and in solving some of the complicated problems of wound healing.

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DISCUSSION OF PAPERS OF DR. BRENIZER AND DR. DAVIS

Fenwick Beekman (New York City): We have used the small, full thickness pinch graft extensively at Bellevue Hospital in the Children's Surgical Service. There we have had a large number of burns, and also a great many cases of avulsion of the skin from the limbs. In all of these, we have used the small full thickness skin graft.

A point to be stressed is the preparation of the area to be grafted. In fresh burns, the grafting should be done just as soon as the eschar of the burn separates from the surface. We find that after this, the use of a wet dressing for twenty-four hours is sufficient, and then the part can be grafted.

In avulsion of the skin, the grafts can be placed as soon as the infection is cleared up. As Dr. Davis has said, the grafts can be used in some areas of the wound before others; the areas in which pockets of infection have been cleared up are ready first.

We so often see neglected burns, burns in which no grafting has been done and in which the epithelium has covered part of the limb but left ulcers. In these cases it is very important that the granulating area be entirely excised. If the excision is not made, there is not sufficient blood supply in the area and the grafts will surely fail.

My experience with isografts has been exactly the same as Dr. Davis', and for a great many years we have never attempted their use.

I think the work of Leo Loeb, done some years ago, is very interesting. He proved then that blood grouping and similar tests were useless, that isografts even from parents and brothers were useless, but that in *like twins*, isografting could be done. Identical twins have similar body reactions.

DONALD M. GLOVER (Cleveland): I was very much interested in Dr. Brenizer's cases of harelip and cleft palate and I would like to ask him concerning the fate of the fascia grafts used. It has been my experience that fascia grafts do not tolerate infection very well and, of course, in the oral cavity it is very difficult to avoid a certain amount of infection.

In regard to Dr. Davis' paper and illustrations showing the excellent results obtained with small deep grafts, I would like to emphasize further a fact which he brought out. It has been embarrassing to all of us to note the scarring of the donor areas. If these areas are not carefully selected or are scattered about the body, an undesirable type of skin remains permanently. All of us have, I think, had the unfortunate experience of having patients come in later life for plastic work upon the face after they have had numerous small full thickness grafts removed from various parts of the body in childhood. At the time we need to have good skin for full thickness grafts it is almost impossible to find an area of clear skin anywhere on the body. Some of us prefer to use the split thickness graft in areas such as Dr. Davis has described. It seems advisable to cover very extensive wounds completely with split thickness grafts (or if the entire area cannot be entirely covered at one sitting, one portion of the denuded area is completely covered, the next area at a subsequent grafting operation, and so on) because granulating areas are not left between grafts as when small deep grafts are used. A patient continues to lose weight and develop secondary anemia as long as sizable granulating areas persist.

In regard to isografts, we have for several years been doing experimental skin grafts in puppies and have found that in young litter-mate puppies we can get very satisfactory cross graft takes. However, beyond the age of about six months we have not been so successful. This is just a preliminary statement that may in the future offer some possibilities.

JOHN A. CALDWELL (Cincinnati): Our experience with the full thickness graft has abundantly corroborated all the claims made by Dr. Davis. It is by all odds the most useful way to restore denuded areas. We have also had absolutely identical experience with the isografts.

A method that we use in the treatment of the donor area is a distinct advance where the area is not too large, as for instance, an area in the thigh. Instead of taking small islands from a wide area, we will take them from a strip, possibly an inch wide, down the front of the thigh and have them so close together that the entire skin area from that strip will be denuded, and be excised and drawn together. A straight line incision results which heals by first intention.

ADDISON G. BRENIZER (closing): Dr. Glover asked about the fate of fascial grafts exposed to infectious cavities, as in the mouth or the nose. On the advice of Dr. Blair during the war, we used strips first of all to help hold the mucous membranes together in gunshot wounds about the face. Some persisted, and some did not.

I mentioned that in using a strip of fascia to take the tension off the stitch line in cleft palate, we had two bony palates left over. Grittiness of the palate and an inability to stick a sharp instrument through the bony part meant bone. Some palates became a little gritty anyhow. I believe, although I did not see the fascia again, that it persisted in spite of exposure to the nasal mucous membrane.

In several cases where the Victor Veau operation was used, the fascia was never seen again and served the purpose of taking the tension off the stitch line. Even ribbon catgut will do that. In some of these cases, we have, after a week or ten days, seen some part of the fascia again; in other cases we have not. Where the fascia was imbedded in the lip, it was not seen again. An objection to its use in the lip is that it produces unnecessary thickness, but this later disappears. We never see that fascia again. In some cases it remains; in others it may act as a scaffold and become absorbed like any foreign material. It serves the purpose of keeping the tension off the stitch line—and that is our requirement.

The first operation on cleft palate and harelip is the best opportunity for permanent repair, because never again is operation quite so easy. The disappointment of the parents if one fails is a further impediment to later attempts.

I have seen a doctor whose daughter was operated on by this method a number of years ago. She had been taken around the country and it was said that her palate could not be closed. She was advised to wear a diaphragm. Fascia was used, however, and although the midline did not quite meet, the fascia persisted until the midline did join. She now has rather a good palate, and they say that she actually sings well.

HIGH TENSION ELECTRIC BURNS*

CHARLES REID EDWARDS, M.D. AND HARRY C. BOWIE, M.D.

BALTIMORE, MARYLAND

I WISH to present this subject entirely from the viewpoint of the clinical surgeon. While much creditable work has been done in the laboratory, conditions prevailing where the workman is exposed to high tension wires or equipment cannot be duplicated there. A combination of circumstances, such as temperature, humidity, dryness or moisture of the skin of the workman, the areas of the body making contact, the size and shape of the electrodes, whatever they may be, duration of contact, and finally voltage and amperage, is difficult to reproduce.

Electropathology has been so well considered by Jaffe that it needs no elaboration. It is well known that electric current kills tissue, and that there is quite a difference in the way in which various tissues react to the passage of electric currents. Bone offers the maximum and skin the next greatest resistance to electric current, and blood is known to be the best conductor of electric current in the body. It naturally follows that the widest destruction of tissue is observed where resistance is greatest. This is of considerable importance and its application is seen in the location and type of burns encountered.

The classification of burns, as usually understood and accepted, really does not hold here. In destruction of tissue due to electric current heat is generated within the tissues, and destruction will follow wherever the current goes, provided it is applied long enough. In the ordinary burn heat is brought to the tissues from without. In high tension burns desiccation and charring occur immediately, beginning at the points of contact and extending along the path pursued by the current. The tissue is so completely devitalized and mummified or charred that there is no absorption from it, and consequently no toxemia. The ordinary first, second, and third degree burns seen in association with electric burns are usually due to the ignition of clothing or some inflammable gas or dust, and, therefore, are not true electric burns. I propose, accordingly, that we discard the old classification when dealing with burns due to electricity, and speak of them only as "electric burns." They are usually the result of a voltage somewhere between 1000 and 4500. Low

voltage, such as lights our homes, the usual 110 or 120 volt house current is seldom responsible for burns, but is frequently responsible for sudden death. Most of the severe cases here considered have been
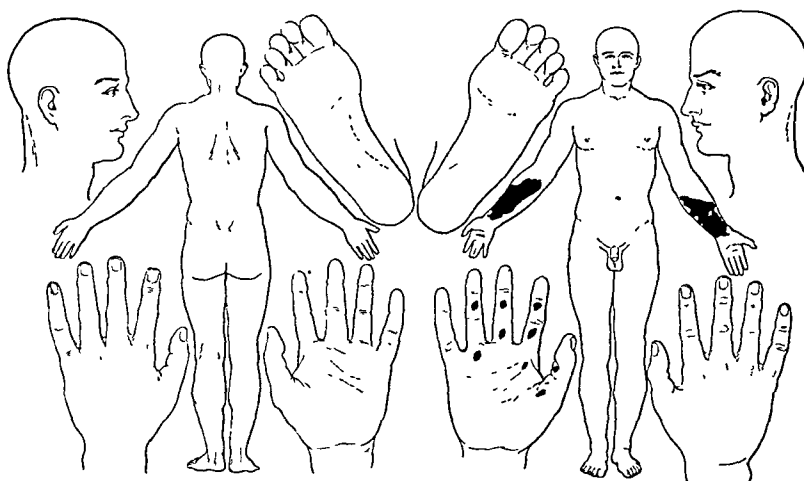
FIG. 1. C. B. Admitted immediatedly following accident in which contact was made with right hand and flexor surface of both forearms. Voltage was 500. The charring was so superficial that in two days it was possible to discharge the patient from the hospital. His recovery was complete.
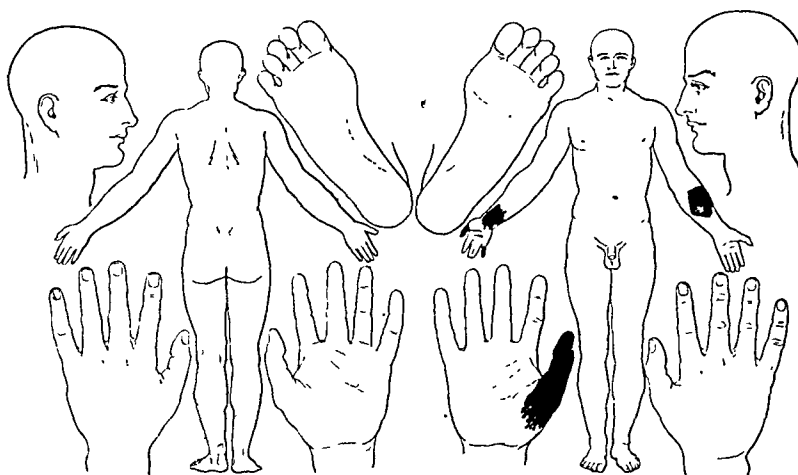
FIG. 2. C. M. Admitted soon after accident in which he made contact at several points involving right hand and wrist and left forearm. The voltage was 2,300. All injured areas were ischemic and soon became mummified, permitting a debridement of burned flat areas and requiring an amputation of the right thumb. The right fifth finger was entirely rigid due to damage to flexor tendons. It was painful and useless, therefore was amputated. A plastic operation permitted the use of the first metacarpal as a thumb.

exposed to less than 4500 volts. Some who were exposed to the higher voltage, 25,000 to 30,000, suffered severe local burns, and one, or
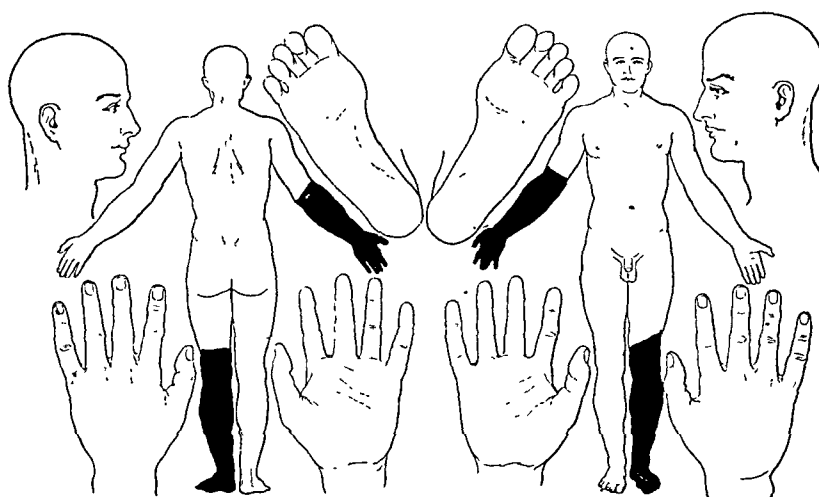
FIG. 3. M. H. Admitted one-half hour after injury, sustained by contacting right hand and left foot against a 4,000 volt line and pole. Contact maintained for one and one-half minutes. There was charring of foot and hand with ischemia and discoloration extending far up each limb. Complete mummification of affected limbs followed. Secondary hemorrhage at line of demarcation, right arm, necessitated a guillotine amputation at mid-arm on third day after admission, and anaerobic gangrene developing at line of demarcation, left thigh, required guillotine amputation of mid-thigh on seventh day. Complete recovery except for loss of limbs.
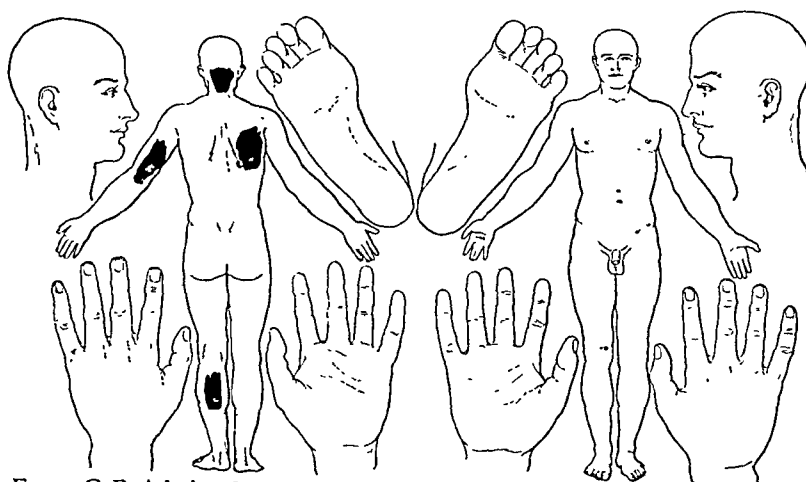
FIG. 4. C. B. Admitted to another hospital July 13, 1922, suffering from a severe burn on back of head, neck, back, left arm and left leg. He had fallen over a high tension wire. He was admitted to the University Hospital August 22, 1922. The wound on the head was badly infected. The outer table of the occiput, an area of 5 by 6 cm., had sequestrated and was removed. The wound was then grafted. The other wounds had healed by granulation. Patient recovered completely, and although badly scarred, he has been at work continuously.
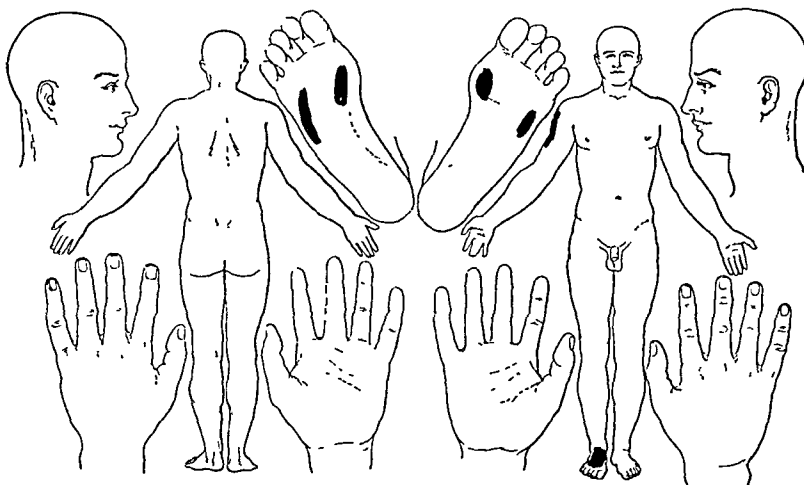
FIG. 5. H. C. While working 10 feet above the floor, contacted wires in unknown manner with right deltoid region and feet. He suffered electric burns of right arm, dorsal surface of right foot, and two areas on the bottom of each foot. The tendons on the dorsal surface of the right foot were exposed when the coagulum was removed twelve days after admission. Superficial areas of necrotic skin were removed from the bottom of the feet, and six weeks after admission skin grafts were applied to the dorsal surface of the right foot. He was discharged about eleven weeks after injury, recovery being complete.
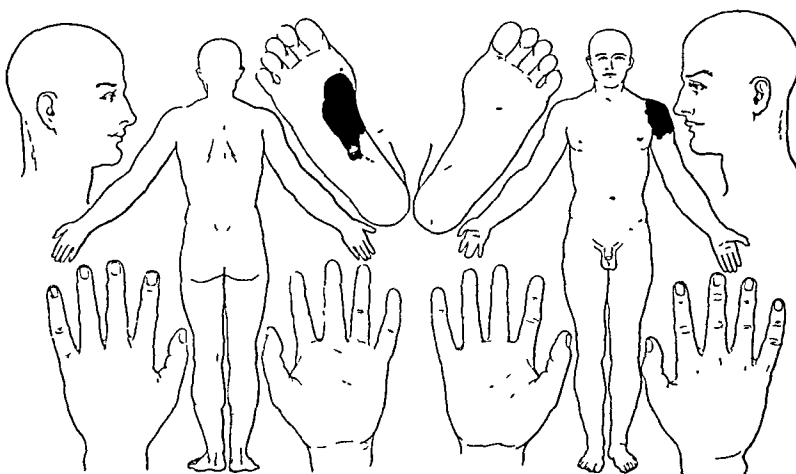
FIG. 6. W. A. Admitted immediately following accident in which left shoulder and right foot were in a short contact with a 13,000 volt wire. Patient dropped to the pavement and suffered an intertrochanteric fracture of the left femur. The burns were deep. The flexor longus hallucis tendon was exposed for 1 inch in the wound on the right foot. The wounds were packed with gauze saturated with azochloramid. Complete recovery followed.
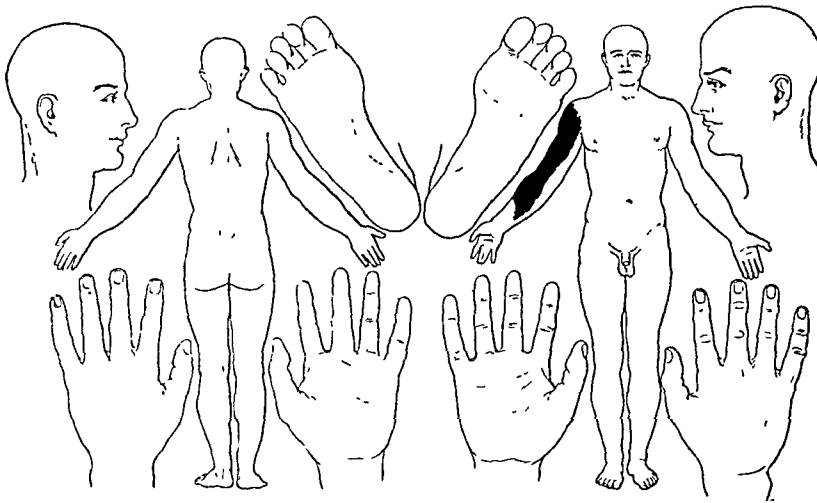
FIG. 7. L. N. Admitted immediately after accident in which he sustained a charred burn of the right forearm and arm. The flash ignited his clothing, and as a result of this he suffered a second degree burn of the trunk. Shock followed. Albumin, red blood cells and casts appeared in the urine, and visual defects, which persisted, for several weeks, were noted. All wounds eventually healed, and recovery was complete.
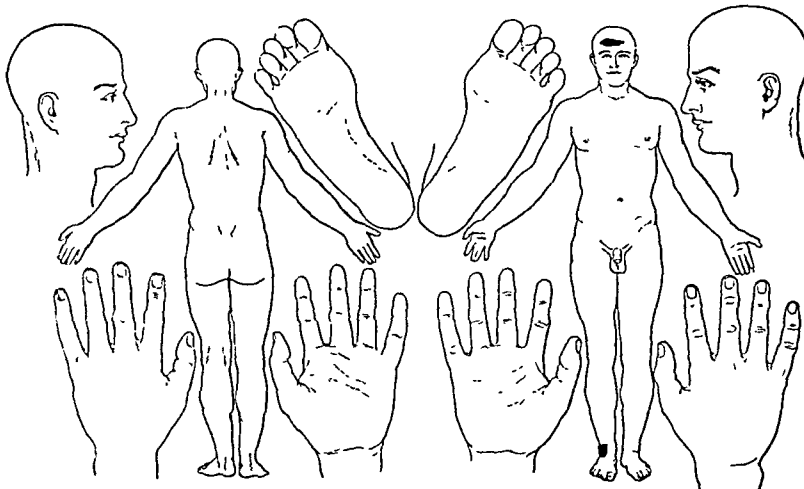
FIG. 8. J. R. Admitted within an hour after the accident in which contacts were made with right ankle and forehead. Second degree burns only were sustained. No complications developed. Observation discontinued after three days, and patient was therefore discharged from hospital. Recovery was complete.
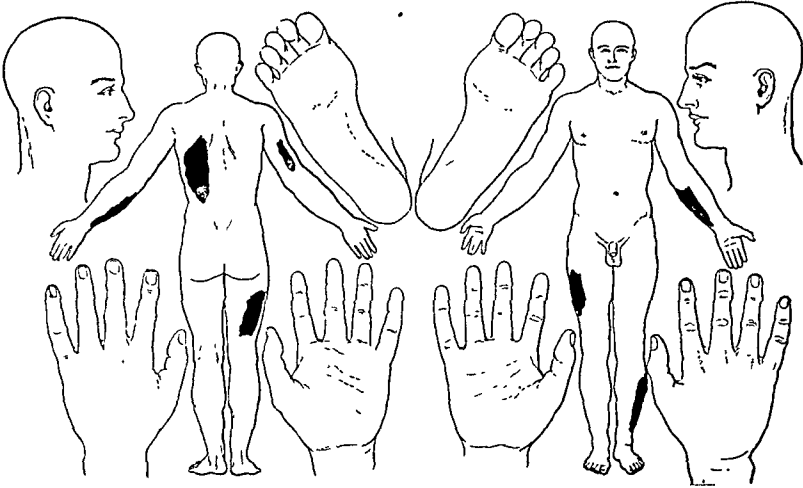
FIG. 9. A. M. Patient suffered deep charring burns of the left forearm, left side of back, and left leg, posterior surface of right thigh, and right arm. The coagulum separated slowly. Wounds were then prepared and skin grafts applied. Patient was discharged from the hospital about ten weeks later. Recovery was complete, and he was able to resume former duties.
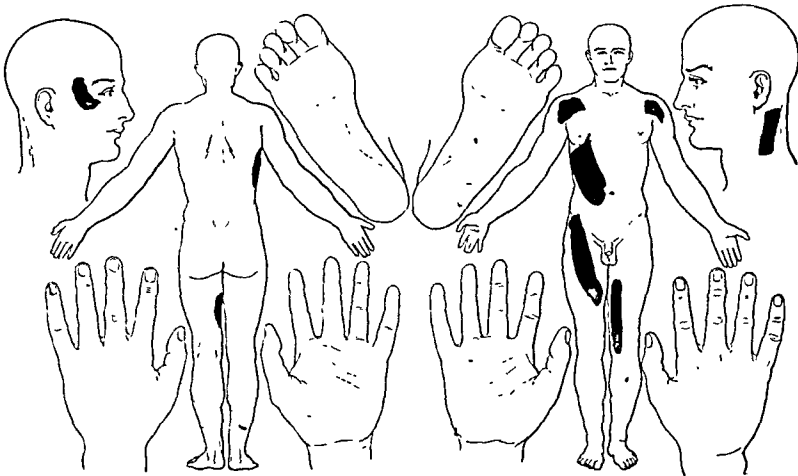
FIG. 10. W. R. Admitted soon after accident in which he fell across a 2,200-volt wire and suffered deep, penetrating, charring burns of the right side of the face, right shoulder, right chest and abdomen, right inguinal region, and anterior surface of right thigh, left shoulder anteriorly, and left thigh internally. All wounds were serious. The soft tissue fell away from the fourth and fifth ribs, and they were removed. The right lung collapsed and a bronchopleural fistula developed. Secondary hemorrhages occurred from the wound over right shoulder and in the right inguinal region. The right zygoma sloughed out, and a cataract developed in the right eye. Patient was very toxic for six months. He remained in the hospital for eleven months, and later returned for skin grafting to chest wound. All wounds eventually healed. After cataract was removed from the right eye, there was little use of the eye. Patient was able to return to lighter duty eighteen months after injury.

possibly two, where an arc developed, exhibited unusual complications. This is probably due to the fact that in an arc the temperature is in excess of 2000°C. and such extensive damage is done that death will almost inevitably follow.
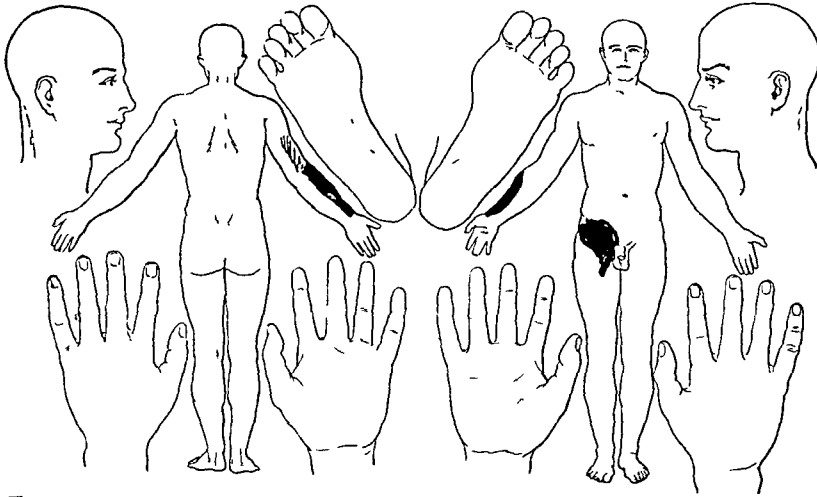
Fig. 11. H. H. Admitted soon after accident. Suffered electric burn of radial side of forearm which extended in a spiral manner to the elbow, and from there the damage was extended up the arm as a second degree burn. There was also an electric burn in the right inguinal region, which extended down the thigh as a second degree burn. The coagulum separated very slowly. A debridement was performed seven weeks later and skin grafts applied. The hand was very badly swollen and some of the radius exposed. The wound was kept clean and finally healed, after some of the extensor tendons had sloughed. While considerable deformity of the hand resulted, the use of it has enabled the patient to be on active duty for over ten years since his accident.

Because of the kind of duties performed by electricians working about high tension wires and equipment, it naturally follows that at least one point of contact will usually be the hand, and frequently it is both hands or hands and feet.

Since, next to bone, skin offers the greatest resistance to electricity, it is a common experience to see severe burns in the hands, and, since hard, calloused skin is more resistant than thin, delicate, loose-textured skin, greater destruction of tissue must be anticipated. The tendons and ligaments come next before the super-resistant bone is encountered, and the rich vascular plexus, the best conductor of electricity within the body, is to be found throughout both the hands and feet. How perfect a set-up, anatomically, for a serious destruction of tissue, once contact has been established!

Charring of tissue is more definite and instantaneous the higher the voltage, up to 5,000 to 10,000 volts. Therefore in the some-

what lower level of high voltage current, damage to tissues, not immediately manifest by desiccation or charring, is frequently encountered. The current penetrates deeply, through skin and under-
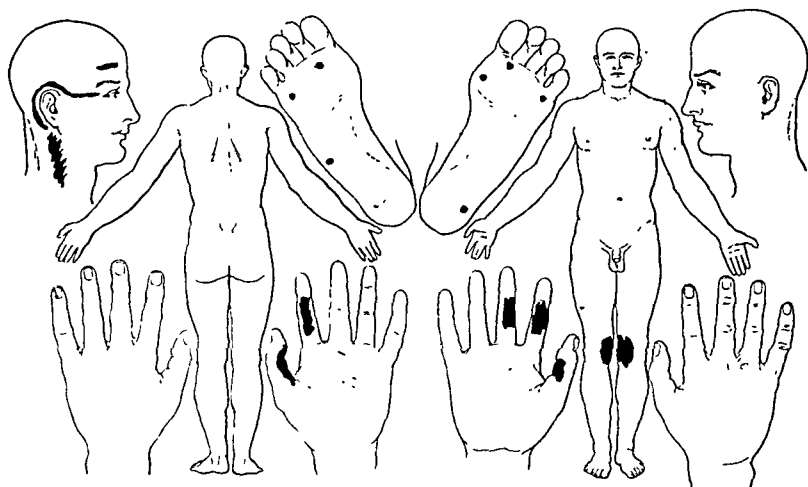
FIG. 12. W. M. Testing at transformer station. Kneeling, and when he removed pliers from right thigh pocket contacted a 30,000 volt line. He was unconscious for about one-half hour. Artificial respiration by companions. On admission there were charred burns over and behind right ear, following glasses shaft, a superficial burn from behind ear to thyroid region, where the current jumped from glasses shaft to zipper on sweater. Ischemia over each inner condyle of both femurs. These areas soon turned black. Conservative treatment employed. Azochloramid dressings twice daily. When slough had separated, one could see through the thin membrane sealing the knee joint. After healthy granulation tissue had developed, skin grafting was employed.

lying structures, often charring only the very superficial layer of skin, but enough damage is done to the tendon sheaths and tendons to set up an inflammation resulting in dense adhesions between these structures. Sometimes what appears to be a superficial injury develops into a very serious and far-reaching one, and quite the reverse is also true.

Burns over joints and tendons are often apparently trivial, but within a few days are found to be quite extensive. If an immediate debridement is undertaken the tendon sheaths and joint spaces will be opened widely, while, if conservative treatment is followed, sufficient granulation will occur to seal these spaces beneath the slowly separating coagulum.

When bone is attacked by high tension current, maximum resistance is encountered, and most of the damage is done to cortical bone. Dead bone slowly separates as the reparative process comes to the rescue, and when sequestration is complete and the seques-

trum has been removed, the area can very soon be prepared for skin grafting.

Various neurologic symptoms have been singularly lacking in this group of cases.
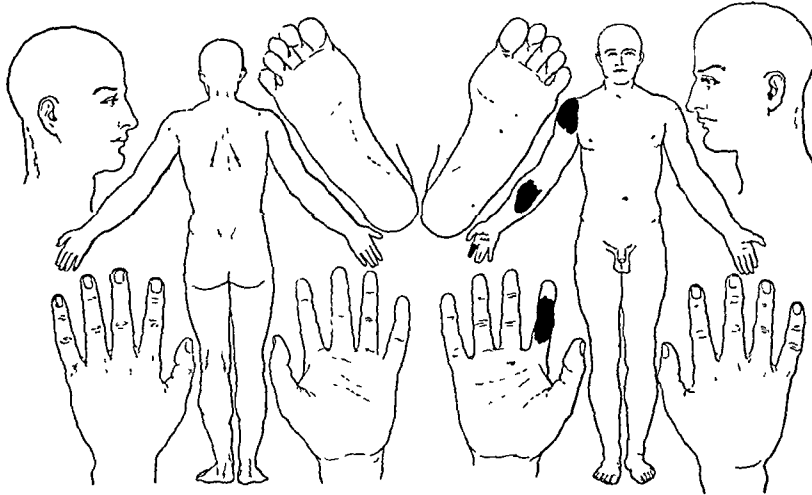
FIG. 13. T. M. Admitted immediately following injury in which contact was made with several places on right upper extremity. A voltage of 33,000 was encountered, resulting in a very superficial cutaneous charring. Recovery quick and complete.
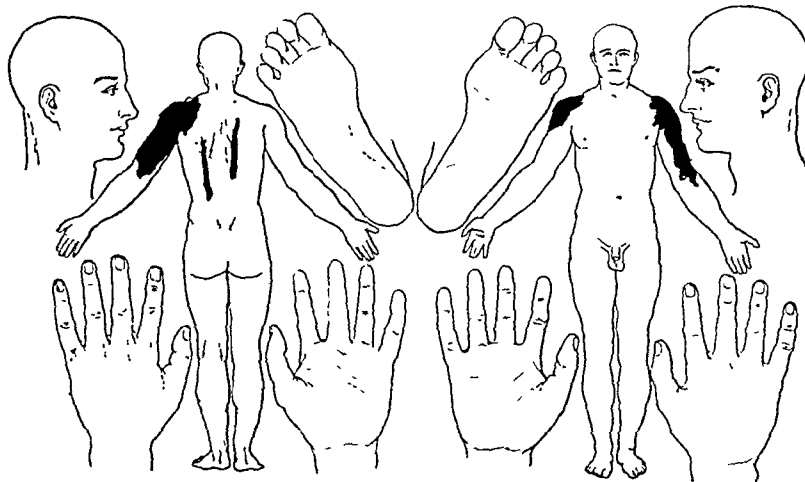
FIG. 14. G. H. Admitted soon after accident in which contact with 4,500 volt line was made with both shoulders and back. Charring followed. The coagulum separated satisfactorily, and one month after the accident skin grafting was done. Recovery was complete.

Treatment of the Local Burn. The surrounding skin is thoroughly cleaned with soap and water wherever possible, and then alcohol compresses are applied to the skin surrounding the coagulum.

When the latter begins to separate and its edges curl up, it is carefully trimmed, removing all loose sections. Then the surrounding skin is covered with vaseline or lanolin and azochloramid in olive
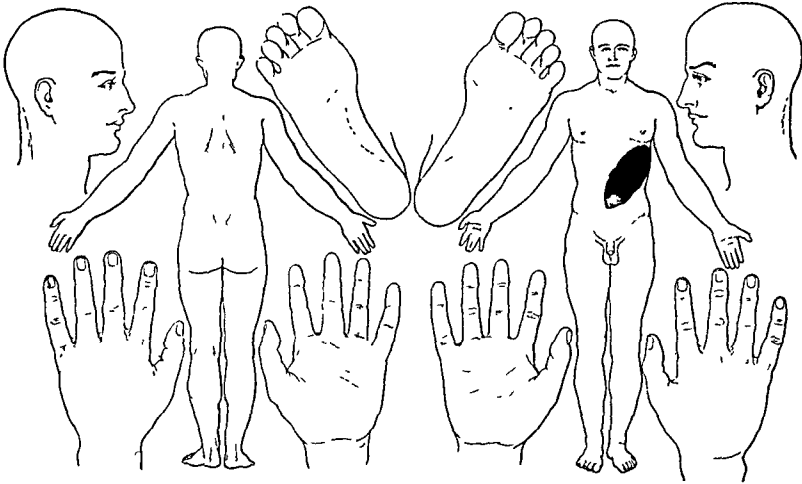
FIG. 15. Leaned against a transformer, and suffered a charred burn of the left side of the chest and abdomen. He remained in the hospital one week. No complications developed and the slough was separating satisfactorily. He was readmitted two weeks later and a debridement was done. Three days later skin grafts were applied. Recovery was complete.
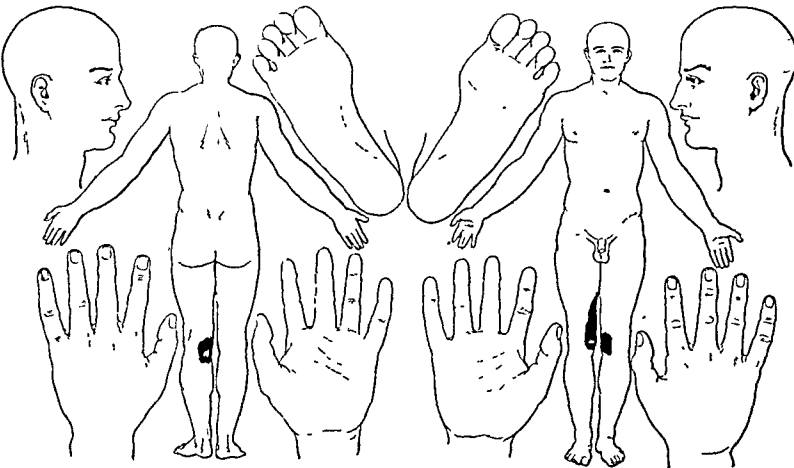
FIG. 16. P. S. Admitted immediately following accident in which contact was made with each thigh. On admission there was an ischemic area over each point of contact. Within three days death of tissue was definite and superficial. An excision of each burn was done, the wounds completely closed, and primary repair followed.

oil is applied to the ulcer. This is repeated until the entire coagulum is removed, and when the base of the ulcer presents uniform healthy granulations, skin grafting is done.
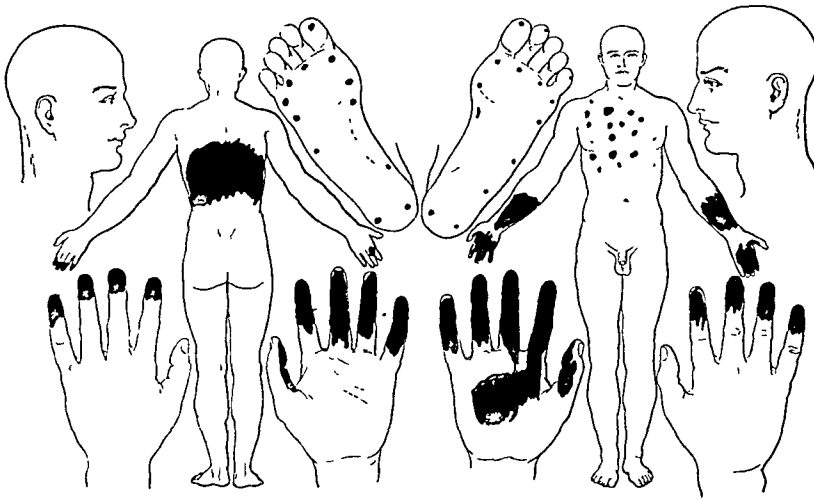
FIG. 17. R. K. Was admitted one-half hour after accident. Conscious and comfortable, but severely burned over hands, right arm, back, and chest. The burns on chest were charred discs leading to sternum and ribs, evidently the result of an arc which had split. The punched-out charred burns on feet corresponded to location of the tacks in his shoes. There was no severe shock, and patient was entirely rational and uncomplaining, exhibiting no paralysis. Seven hours later coma suddenly developed and death followed in one-half hour.
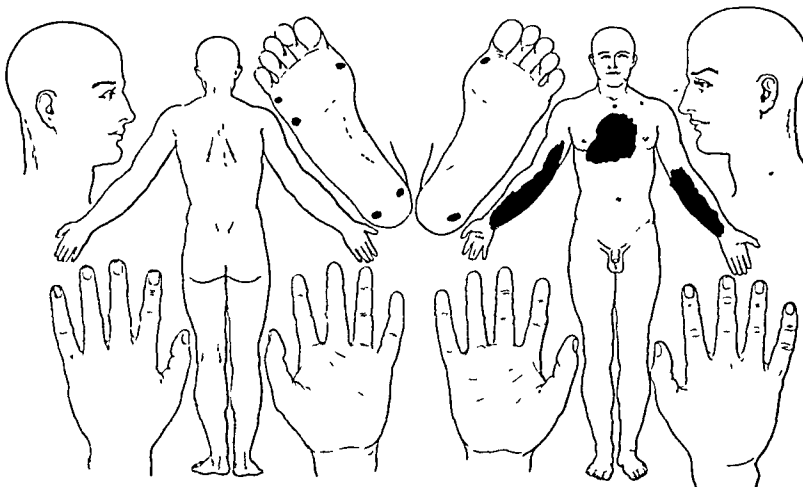
FIG. 18. H. S. Admitted immediately after an accident in which patient attempted to throw open a switch. There was a blinding flash, and he suffered an extensive burn over anterior chest wall, both arms, and penetrating charred burns on the plantar surfaces of his feet, these latter burns corresponding to the location of the tacks in his shoes. His chief complaint was tightness in the chest. Three hours later he expectorated bloody mucus, and developed a nephritis. The voltage was 26,000. His chest distress increased and death followed.

The charts illustrate the location and size of burned areas, and a short description of each case is appended. The shaded areas represent actual electric burns. Where definitely known, the voltage is stated.

CONCLUSIONS

1. Destruction of tissue due to high tension electric burns should be considered as electric burns.

2. The customary classification of burns should not be applied to those due to electric currents.

3. Immediate or early operations, such as debridement, should be employed only in the superficial, often linear type of burn, on the back or upper portion of extremities.

4. The conservative method of permitting nature to build up new tissue beneath or around the destroyed tissue leads to the most satisfactory ultimate results.

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DISCUSSION

FENWICK BEEKMAN (New York City): All I can do is congratulate Dr. Edwards on his admirable paper. I do not think that any of us have had the experience he has had in electric burns. Mine has been very limited. I have had experience with burns, but little with electric burns. I hardly feel competent to discuss this excellent paper. I am sure I could add nothing, but might take something away. I congratulate Dr. Edwards on his presentation.

CASPER F. HEGNER (Denver): Dr Edwards mentioned the obscure internal effects of high tension burns. They are extremely interesting, certainly for me, for I stand here as a man who was once electrocuted. I was setting a fracture of the leg under the fluoroscope. The patient lay on the table, and around his ankle was a clove hitch with the ends around my back. I pulled on the leg and told the assistant at the head of the table

to pull the patient up. In doing so, he pulled me with the patient. There was a short circuit, and I was knocked out. My only sensation was as if a mule had kicked me in the chest. My knees were blistered, my hands were blistered, and over the heart area my shirt was burned. I was picked up from the floor and was able to continue the fracture reduction, but for about three months thereafter I had a peculiar cardiac insufficiency.

In a little experience with lightning burns, I have found that they may not leave any evidence of burns on the skin, as Dr. Edwards has shown, but they do leave cardiovascular changes. I should like to ask Dr. Edwards whether he has had any experience with the cardiovascular reactions in high tension accidents.

CHARLES REID EDWARDS (closing): I want to thank the gentlemen for their comments.

Our conservative treatment is as follows: To see that the surrounding skin is thoroughly cleaned with soap and water, dried with ether and then alcohol. Compresses are kept on the surrounding skin, certainly for twenty-four or forty-eight hours, and then sterile dry dressings until the coagulum begins to separate. At this point we cover the surrounding normal skin with vaseline or lanolin and apply azochloramid in triacetin, or olive oil. We prefer the olive oil. Dressings are repeated every day. The coagulum is permitted to curl, and then the curled, loosened edge is removed as rapidly as possible.

The cardiovascular symptoms, strange to relate, while readily produced in the laboratory and reported by most men writing on electric or lightning burns, have not been observed in any of our patients, even though they have been very seriously burned.

Of the eighteen patients that I have reported, two died. These were the patients subjected to the arc type of current and apparently there were pulmonary complications.

I did not go into a discussion of the electrical phenomena because much has been written on that. I can refer you to the bibliography at the end of my article.

The pathology has been ably reported by Jaffe and I see no necessity for further elaboration. I am simply calling your attention in this presentation to a very interesting and hazardous type of burn.

THE RELATION OF TRAUMA TO INGUINAL HERNIA

AN ANALYSIS OF 1,376 HERNIOTOMIES

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E LSEWHERE in previous publications[1-5] I have attempted to express my views on this subject. My attempt today is to analyze the operative findings in which trauma was the alleged producing factor in inguinal hernia.

The central theme is to prove statistically that isolated trauma, a single act of violence, is never the sole producing cause; further, that trauma is an aggravating factor only when and if certain criteria obtain.

Let it be understood that the subject under discussion is inguinal hernia in male adults and the relation trauma bears to the production of this lesion. There is, of course, nothing new in this inquiry, for the relationship of trauma to hernia has long been a vexing and perplexing problem and there is a vast literature on the subject, medicosurgical as well as medicolegal.

The problem is twofold. The first is the question: "Can hernia be caused by trauma?" The second the question "If hernia is not caused by trauma, can it be aggravated thereby?" In other words, we seek to determine: (1) causation, and (2) aggravation.

1. *Causation.* Can one act of trauma become the sole producing factor in the development of a hernia? This question is scarcely controversial because there is now general agreement that no single, isolated or individual act of trauma is causative in a normal individual unless there is actual laceration of the overlying structures. This virtually implies incisional hernia, compound or open wound hernia, if you prefer, and that sequence of injury is so rare as to be negligible.

It is universally conceded that a hernia does not and cannot develop in the absence of a preformed sac, and to that degree hernia is born and not made. In other words, there is a congenital predisposition or an acquired anatomic defect leading to the formation of the peritoneal pouch, the diverticulum of peritoneum that we call the sac. In the absence of this peritoneal irregularity, this bulging of the visceral covering, there can be no protrusion of it

through potential openings and hence, there can be no herniation. This doctrine of a preformed sac is established by abundant anatomic and surgical proof. There is no controversy in answering question 1 by stating that trauma is unrelated to the causation of hernia in the absence of demonstrable laceration of the overlying structures. No amount of cross examination can shake this fundamental assertion.

2. *Aggravation.* Can trauma aggravate, accelerate, accentuate, increase, modify, hernia? The answer is "Yes," if the attending circumstances so indicate.

What are these "attending circumstances"? There will be no controverting the assertion that the main essential is increase of intra-abdominal pressure. Only one other element, in the absence of penetration, could be equally productive, and that would be deliberate or purposeful dilatation of the external ring, the inguinal canal or the internal ring. That very procedure is still accomplished by professionals in certain countries where military service is obligatory and where the presence of hernia is sufficient cause for rejection.

But that contingency does not interest us and it is mentioned only to show how difficult and painful it is to interfere with these well-guarded openings by attempting suddenly digitally or instrumentally to dilate them from without. In passing, let it be stated that these professional dilators often make three attempts before they are successful, and in the process they inflict enough pain to cause the victim to faint. The suggestion and implication is that if deliberate dilatation is so painful and disabling in a prepared and desirous subject, how much more painful must be the process in an unprepared subject? In other words, sudden onset by unexpected force must, in accredited cases, be accompanied by pain and other symptoms.

If intra-abdominal pressure is the prerequisite, we can postulate our problem by the statement that this pressure must be adequate in degree and adequate in effect. Hence, there must be sufficient force to register in terms of sudden increase of intra-abdominal pressure in order to produce dilatation of a ring, or to produce a protrusion between the rings. There can be no hernia unless there is dilatation of a ring, of the canal, or of muscle and fascial coverings.

Elsewhere[4] I have called attention to the resemblance of the birth canal to the inguinal canal, the internal os resembling the internal ring, the external os the external ring, and the cervical canal resembling the inguinal canal. If impregnation and growth

occur, then intra-abdominal pressure in the one case produces the fetus, and in the other, the hernia. But the prerequisite in each case is impregnation, growth and intra-abdominal pressure.

An obstetrical analogy of this sort is quite apt and no one familiar with the subject would say that the last pain produced the fetus, although some doctrines regard the "last pain" as the producer of the hernia.

Then, if intra-abdominal pressure is the essential, what constitutes force enough to be regarded as actually causative? This can be summated in terms of *adequacy* as to site and *adequacy* as to effect. A light blow on the upper abdomen should be less productive than a heavy blow on the lower abdomen. A distant blow, a wrench, a sudden strain, a violent cough, a valiant sneeze, straining at stool, extreme vomiting, lifting, pushing up or pushing against—all these, singly or together, have been accused as causative. The relationship of any or all of them has often been so apparent that practically all the laity, and many of our profession, believe that the cause and effect are indisputable.

Let us pursue this idea still further. It is certain that many individuals are so built that, sooner or later, a hernia will develop. This we know because of laxity of the abdominal wall, paucity of muscular and fascial supports, clefts or apertures in muscle or fascia, associated varix, and a variety of congenital or acquired defects which weaken this anatomic section. Thus regarded, hernia is a ptosis. Further, we know that many persons have hernia and know nothing about it. As proof of this, the Surgeon-General's office informed me that of 2,754,000 men examined for duty in the Great War, 4 per cent between the ages of 21 and 30 had a hernia well enough developed to cause rejection for military service. If this is true in men less than 30 years of age, how many more must there be in the decades beyond 30? Further, one examination may not be enough, for we all know that the sac may, at times, be unoccupied by intestine or omentum and thus escape detection, and yet at another time become perfectly apparent. Many times I have had patients referred to me by a competent examiner who diagnosed hernia, and yet, despite all sorts of straining, jumping, pushing and coughing, it has been impossible for me to demonstrate the protrusion stated to have been palpable or visible, or both. The sac may be there, but, unless filled, it escapes detection. To be effective as a producing factor, the intra-abdominal pressure must be adequate to fill the sac and perhaps propel it sufficiently to bring it into view or into palpatory reach.

To carry out the obstetrical analogy, it must become a dilating agency sufficient to make apparent the presenting part. Hence, if this element of *adequacy* prevails, it needs no argument to sustain the view that the *effects* of this pressure must also be adequate.

In any form of trauma there is no incubation period; the cause is immediately succeeded by the effect. The sudden onset produces the sudden effect, and there is no interval or waiting period in any traumapathy.

What are these effects? *Pain,* obviously, is the first and this should register enough and be sufficiently disabling to make the possessor aware of it at once. Even under local anesthesia in a patient dulled by morphine or barbital, we cannot at operation handle the sac or its contents without producing pain. If we cannot do it then, how is it possible for a hernial sac of recognizable size to appear without pain? And if the pain is not disabling, or is transient, and unaccompanied by nausea, imminent syncope, testicular dolor and other appropriate signs, are or are we not justified in saying that the traumatic act in question was incidental and not productive? The answer is self-evident, unless we are willing to scrap all we thought we knew surgically and anatomically of this section of the body.

If a man comes to me the day after he says he received his hernia by a lifting strain, and he tells me he had some pain, but not much, and he kept on working, and that night he "found a lump in the groin," can I, as an examining surgeon, expect to find clinical proofs to link the "strain" with the "lump?" Yes. What are the proofs? *Swelling* is one, perhaps *ecchymosis. Local tenderness* is another. Inability painlessly to palpate the protrusion, or to introduce the finger into the ring are other signs, for we all know so richly endowed a section will not fail to resent our examination in terms of pain and tenderness. What am I seeking to determine? Actually, the patient must have sustained an acute peritonitis or diverticulitis if this asserted "strain" bulged into the preëxisting sac to the extent claimed. Now, no medical man within hearing will assert that a segment of peritoneum can be violently thrust from its normal habitat without resenting it unmistakably, and announcing it by appropriate signs and symptoms.

Let us go on and operate upon this patient, note the findings, make a biopsy and send the sac to the laboratory.

With the patient under spinal anesthesia the modified Bassini or Ferguson operation is done. Assuredly, if the condition is recent, a week old or less, one should find corroborative evidence, such as

exudate, organized hemorrhage, traumatized muscle or fascia, and an inflamed sac that has been separated from the contiguous structures of the cord. I have operated on several so-called traumatic herniae as early as a few hours after the alleged onset, but never have I found the slightest evidence of recent pathology. The thick or thin sac was adherent, it had to be dissected, the contents were often shaped or moulded, suggesting long occupancy, the base of the sac was wide and easily dilated. Frequently the contents were attached by strong bands and in every respect, *aside from the history*, there was not a single vestige of surgical proof to indicate anything recent. Then the sac was sent to the laboratory and shortly the report came back, disclosing chronic peritonitis and fibrosis of a grade that would take a year or more of friction and circulatory reëstablishment to accomplish.

This sequence does not occur occasionally, but it is the persistent rule, the inevitable chain of events. Therefore I long ago gave up the idea that hernia was connected with injury except under the most unusual circumstances.

My assistant, Dr. Louis R. Slattery, has analyzed 1,376 herniotomies performed by me, and I am indebted to him for this laborious survey, the details of which were published.[3]

As to operations, we may summarize our experience as follows:

1. We do not transplant the cord.
2. We do not use fascial transplants.
3. We favor spinal anesthesia.
4. We enforce deep breathing exercises when the patient is out of anesthesia.
5. We do not permit catheterization.
6. We regularly get the patient out of bed on the eighth or ninth day to prevent disuse muscular atrophy.
7. We permit return to light work two weeks after the patient leaves the hospital, and two weeks thereafter he is returned to regular work.
8. We do not believe in the efficacy of the injection treatment except in carefully selected, coöperative patients under the care of experienced surgeons.

Thus far we have alluded to the anatomic fallacy of believing that hernia could by one act of trauma assert itself, and we have also referred to the biopsy evidences at operation, and also stated the pathologist's findings. Thus, we have arrayed the anatomist, the clinician, the surgeon and the pathologist in agreement.

Are there further proofs to add to this disclaimer? Yes—and these are from the book of experience, the great teacher.

I do not think we are wrong if we estimate that 10 per cent of our male patients in the decades 20 to 70 have actual hernia, and we know, statistically, as stated, that 4 per cent in the 20 to 30 decade have the condition to an easily recognizable degree. If this is so, then a very considerable proportion of your patients and mine who are treated for other injuries have actual or demonstrable hernia.

Granting this, the inference is that with violence enough to fracture the pelvis, the ribs or the femur, there should be an occasional instance of aggravation of an already existing hernia.

Have you ever been called upon to treat such an aggravation as a concomitant of injuries that inevitably must have produced extreme intra-abdominal pressure? We have all seen crushing injuries of the abdomen and of the chest, with and without intra-abdominal and intrathoracic damage, but I am unaware of any case reports indicating aggravation of hernia. To me, this is one of the very real proofs that aggravation, even under extreme violence, is not a very active factor; and if violence sufficient to rupture viscera and fracture a pelvis or hip does not cause aggravation, it is not surgically logical to ascribe aggravation to lesser causes. Personally, I have never had a patient who required attention for hernia in the presence of associated injury contiguous to or distant from the inguinal regions.

Another point seems worthy of note, and that is the infrequency of strangulation of a hernia under the influence of strain or injury. The majority of my cases of strangulated hernia have occurred at night or outside working hours; and, incidentally, strangulated hernia seems less common than hitherto. If aggravation due to violence, was an important factor, then assuredly strangulation as a sequel should be more common.

However, we are accosted by the clinical fact that pain does occur after some definite act or incident associated with intra-abdominal pressure; and that, in some cases, a lump appears within a few hours or a few days. What causes the pain and what causes the lump? *Pain* can be due to tugging or pressure on the sac, to gaseous distention of the intestine within the sac, to rotation or twisting of the omentum in the sac, to pulling upon the adherent contents of the sac, to irritation by pressure upon the iliohypogastric or ilio-inguinal nerve supply, to muscular strain, and perhaps also to torsion of the cord.

The *lump* appears because the sac is occupied by intestine or omentum, or both, and it then acts like a Barnes or Voorhees obstetrical bag to dilate the orifice or the canal—virtually a prolapse. But, as stated, neither the pain nor the lump denotes recent origin unless corroborated by local tenderness, edema and perhaps ecchymosis.

Hernia is not actually an acute surgical entity, but, in reality, a progressive disease characterized by periods of remission and exacerbation. Inevitably, the lump will appear if the already mentioned factors pertain, and sooner or later, peristalsis will balloon the sac sufficiently to make of it a dilating agency. The accused intra-abdominal strain or injury may be the *ascribed* factor in the opinion of the patient, but the *ascribable* factor is the normal development of a progressive disease, manifesting itself by an exacerbation.

As physicians, we often have to differentiate as between the *ascribed* and the *ascribable* cause. A patient may *ascribe* his attack of abdominal cramps to a fish dinner, but any physician would assert that the symptoms were *ascribable* to an exacerbation of gallstones or appendicitis. Assertion is not proof; the *ascribed* is not always the *ascribable* in any intra-abdominal lesion, hernia not excepted.

In differentiating, then, as between an old and a new hernia, there are certain criteria, certain proofs based on professional knowledge, to which we should give heed. (Table 1.)

TABLE 1
CRITERIA

	New Hernia (Acute)	Old Hernia (Chronic)
1. Type of injury	Intra-abdominal pressure from contiguous impact	Ditto from remote impact
2. Type of person	Muscular	Non-muscular; ptotic build; fat
3. Time of onset	Immediate	Late
4. Type of symptoms	Pain; nausea; tenderness; edema	Slight, if any
5. Physical findings	Tenderness; swelling; ecchymosis	Absent or slight
6. Operative findings	Hemorrhage; edema; no adhesions; no diastasis, slits or lipoma	Adhesions; extra- and intra-saccular diastasis; slits and lipoma
7. Pathological findings	Acute peritonitis; hemorrhage	Chronic peritonitis; fibrosis

All this has a medicolegal bearing and we often are beset with doubt in attempting properly to allot responsibility. The compensa-

tion laws in some states require definite surgical proof before making an award, and the seven criteria named above are some of the main factors in the determination. In most industries, a preëmployment examination is required and then the applicant is rated as to hernia in some such terms as:

First degree hernia—External ring admits the tip of the index finger.

Second degree hernia—Ditto, plus impulse, plus gaping ring.

Third degree hernia—Ditto, plus lump.

Fourth degree hernia—Ditto, plus lump below external ring.

The question of repeated violence, successive intra-abdominal pressure, and continuous or intermittent strain is, of course, an important element in those predisposed, and in this group it is only a question of time until the process assumes recognizable proportions.

It is surprising, however, to note the frequency of obvious hernia without the possessor being aware of it. Once attention is called to the finding the almost inevitable comment is: "I must have strained myself"; or "I remember lifting something a short time ago."

Repeated intra-abdominal pressure is sometimes an accredited causative factor. Incidentally, under spinal anesthesia, with the canal opened, it is interesting and instructive to ask the patient to cough or strain. In so doing, the sac is ballooned in the same way as if the patient becomes nauseated during the operation.

My conception of the development of hernia is that the preformed sac enlarges. It then acts as a dilating wedge and at the appointed time presents itself as a completed product, palpable, and perhaps visible to the physician and to the patient. That it may occur as the outcome of a single or multiple traumatic agency is not to be decided until the extent of the intra-abdominal·increase is estimated in terms of *adequacy;* if adequate, then the ensuing symptoms are immediate and demonstrable.

In passing, it is pertinent to say that the surgical cure of hernia has also a bearing on its causation. No experienced operating surgeon has failed to note the extensive pathology involved, the nature and diversity of which required years, and not days or weeks, to produce. Further, there is often a congenital absence of the normal protective barriers, and we oftentimes note a wide inguinal triangle or a very thin or imperfect conjoined tendon. Again, other cases show wide separation in the fibers of the internal oblique and definite slits or thinning of the external oblique fascia so that it looks like bacon with the muscle appearing under the fascial aper-

tures. Not infrequently varicosities of the cord act as the dilating agency, to say nothing of that large group in which lipoma of the cord may act as a wedge to force the contiguous structures apart. Obviously, none of these findings can be said to be in consonance with any recent occurrence, for all of them are congenital or slowly acquired.

It has been said that direct hernia is more likely to be acute than indirect hernia, and that scrotal hernia is the patriarch, the ancient of ancients. Bilateral hernia assuredly is practically never acute, and about 60 per cent of my patients have had hernia on both sides. Furthermore, a direct and indirect hernia on the same side scarcely can be called acute, for to produce such a sequence by one act of violence implies great force and inevitable great effect, in terms of disabling pain that should send the patient into a state of shock.

It is a surgical fallacy to believe or to assert that any single act of trauma can, without symptoms, bulge the sac and propel it through normal openings or along the canal in an area so richly endowed with a vascular or neural supply, and so well buttressed by strong muscles and fascia. The sac is a diverticulum and when it suddenly bulges, it may be regarded as producing an acute diverticulitis. If so, it registers like any other diverticulitis, namely, by pain, swelling and tenderness. The appendix is the best known diverticulum and the cardinal symptoms of appendicitis are the well known trinity of pain, swelling and tenderness—just as in "hernia-itis." I am aware that "traumatic appendicitis" is also an interested party in medicolegal controversies, but I have never seen it at the bedside. It has about as much standing as "traumatic tonsillitis" due to hanging or a blow on the neck.

SUMMARY

1. Hernia is never *caused* by injury; the preformed sac is always an antecedent.

2. Hernia can be *aggravated* by injury if the source and symptoms are *adequate.*

3. Immediate disabling pain is the chief symptom, and this is associated with nausea, tenderness, swelling and other manifestations resembling peritoneal shock.

4. At operation, the findings usually denote an ancient process, as indicated by adhesions, extrasaccular and intrasaccular.

5. The pathologic examination of the sac demonstrates chronic peritonitis and fibrosis.

6. Hernia is a chronic progressive disease, a ptosis, a diverticulum, and not an acute surgical entity except in very rare instances.

7. A large proportion of our adult male population have hernia and do not know it; and we, as surgeons, do not know it either, when we are treating them for contiguous injury grave enough to cause aggravation if trauma is a common accelerating factor.

8. Like osteomyelitis, once a hernia always a hernia, and it is subject to periods of accession and remission.

9. If trauma is to be accused and found guilty, then there is always plenty of reliable evidence. This need never be medicolegally seductive nor speculative, but rather, medicosurgically deductive and positive.

10. Trauma has burdens enough without being saddled with this ancient heritage which modern traumatology should cast into the discard along with grape seeds as a cause of appendicitis.

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DISCUSSION

PHILIP H. KREUSCHER (Chicago): The Compensation Act in the State of Illinois provides that before any employee can claim a traumatic hernia he must prove beyond a shadow of doubt that there was no preëxisting hernia. He must also prove that there has been a trauma. Furthermore he must produce evidence of trauma, such as discoloration, swelling, ecchymosis, pain and immediate incapacity.

Despite all of this, the arbitrators and commissioners in Illinois still give compensation to our employees when they have hernias, and in my opinion base their judgment on the clinical history alone. I have in mind one patient in particular who claimed a hernia from lifting. However, according to the clinical story, he was lying under an automobile with both his thighs flexed on his abdomen and in that way was pushing upward on some portion of the car. It is difficult for me to conceive how a hernia could possibly occur under these conditions since the pressure of the thighs upon the inguinal area would, it seems to me, make it impossible for herniation to take place.

In the Carnegie Illinois Steel Corporation we offer an operation free of cost to our employees without actually accepting any responsibility for the occurrence of the hernia.

FRANK P. STRICKLER (Louisville): Dr. Moorhead has had a very rich and extensive experience on this subject, and his paper is excellent. After

reading this paper, one could hardly doubt that hernias are of congenital origin and that trauma aggravates an existing hernia. Any difference of opinion I have with Dr. Moorhead is trivial and does not amount to much.

We have a very happy situation in Louisville. Most of our laboring men are American citizens, employed by American citizens, and when a hernia occurs, it is usually taken care of. The medicolegal aspect rarely enters into the situation. The man goes to the hospital, a surgeon fixes the hernia, and that is the end of it. The insurance company pays for it. I think this is done because it is easier to repair the hernia than to fight the case.

We always transplant the cord. Very frequently a very thick, heavy cord is encountered and it is, in my opinion, necessary to transplant. Better union results when we do it. If the cord is very thick and heavy, we occasionally transplant it externally to the aponeurosis of the external oblique just under the skin.

As anesthetic we use cyclopropane. We usually use kangaroo tendon as suture material, a technique taught by the late Dr. W. B. Coley. I see no reason to change it, although I understand they are now using silk at the Hospital for Ruptured and Crippled. In some cases we use fascial sutures, taken from the external oblique or sometimes from the fascia lata. In using the latter, we always expose the fascia lata and take our sutures under direct vision, because we have had some bad results with fascia strippers. Sometimes there is hemorrhage or a big hematoma after using them.

We are a little more conservative than Dr. Moorhead, in that we keep our patients in bed about two weeks and let them go back to work about a month later. Complications we have had have usually ben pulmonary infarcts, and, occasionally, infection.

I mention the injection method of treatment only to say that, in my opinion, it is not what it is supposed to be. I do not approve of it.

Dr. Moorhead has written an excellent paper; the points I have mentioned are merely pertinent reflections.

ARTHUR R. METZ (Chicago): Dr. Moorhead has well stated the facts on the subject of traumatic inguinal hernia. Any surgeon with extensive experience will agree with the views that he has expressed.

In our experience, involving over 1500 cases of inguinal hernia, we have never had occasion to operate in a case of traumatic hernia. There have been injuries to the abdomen which resulted in rupture to the stomach, intestines, liver, spleen, kidneys and bladder, but in none of these cases has a hernia been produced as a complication. If a traumatic hernia can be produced it certainly ought to develop in connection with these more serious injuries to the abdomen.

In a careful survey of 12,300 men who are actively working, it was found that 6.4 per cent have inguinal herniae. In the same group of men another 7.6 per cent have large inguinal rings and are potential hernia victims. These two classes total 14 per cent of employees in this group who are

possible subjects for a hernia operation at some future date. Only 1 per cent have been operated on for hernia; the others are doing their normal work and are not considering operation.

Everyone interested in the subject of inguinal hernia should review Dr. Moorhead's paper, after which the alleged "traumatic inguinal hernia" will be dropped as a surgical term and will be known in its true state as a congenital inguinal hernia.

EDGAR GILCREEST (San Francisco): We can have nothing but praise for this scientific paper from the anatomical and embryonic viewpoint. We are probably all in thorough agreement with Dr. Moorhead's conclusion that hernia is congenital, but there is a practical point which I think should be emphasized. One thousand bookkeepers will not have so many hernias as one hundred longshoremen or brakemen; therefore, we must concede that the type of work these men are doing plays an important rôle in the causation of the disability. As practical surgeons, we must answer this question in cases of litigation. Therefore, if we admit, as Dr. Moorhead does, that trauma aggravates a preëxisting condition, this, after all, is the salient point. If we make this clear, industry will rely upon the surgeon to say which men to reject and which to employ. When industry hires workers, I think it takes them for better or worse. When men are examined before they are put to work, the surgeon may decide which men have hernia and which have not. The question of traumatic hernia will be relegated to bygone days.

JAMES A. JACKSON (Madison, Wisconsin): I think that most surgeons of trauma are agreed on the point that there is not such a thing as traumatic hernia. However, how much does this avail us if the Industrial Commissions in certain states hold that there is?

In the State of Wisconsin many years ago, I testified that there was no such thing as a traumatic hernia. However, the case was decided against me and they had a rather unique explanation. The Commissioner admitted that the man had a sac previously, although he had never felt any bulging. At this stage it was a potential hernia, he ruled, which had become a traumatic hernia when the content was forced down. Therefore the company had to pay for it.

This was a new conception to me, but the ruling has been followed in case after case.

I think it would be well if some of the Commissions could have Dr. Moorhead's splendid paper read to them; they might change their ideas on it.

Speaking of the injection method of treatment—there are in our Clinic three Jackson brothers. My younger brother, Arnold, has used injections in some 600 cases of hernia. He is very fair about his statistics and thinks his results so far, over a period of about five years, compare very favorably with the operative treatment. My older brother and myself are not entirely

convinced of the efficacy of the injection treatment—the only trouble is that we do not get hernias to operate any more; the younger brother injects them all.

D. C. PATTERSON (Bridgeport, Conneticut): I was very much interested in Dr. Moorhead's paper. I fully agree with him in every point he makes, except perhaps in the non-transplanting of the cord.

I had mixed feelings when I heard Dr. Strickler state that they had no trouble in Kentucky—"they fixed the hernias and that was the end of it." We occasionally get recurrences.

In regard to the injection treatment of hernia, for the last three years I have been interested in this method, principally to determine for myself whether or not the procedure had any value. It has been hard to credit the statistics that have come from the west; in the East we have been slow to adopt the method. Statistics have run from 4 per cent to 98 per cent of cures, and such figures are too fantastic to credit. The injection treatment has, I believe, a small place in surgery, and surgeons should undertake its administration. No one unfamiliar with the anatomy and pathology of hernia should attempt the injections.

I have used the injection method only in simple, indirect, inguinal hernias. I cannot conceive of its application in the average direct hernia. I would not think of injecting a femoral hernia, because it is impossible to tell whether the contents of the sac are reduced, and it is almost impossible to get a truss to hold a femoral hernia.

Some of our patients have been at hard labor for over two years since their injections and are now free from hernia; others have failed to get closure. Why anyone would want to undergo the trouble and nuisance of the injection treatment I do not understand, because it means wearing a truss day and night for at least two months, and then only during the day for the next five or six months. The injections are not always painless. We have never had any severe complications, but in quite a few cases the patient has complained of pain. The advantages of the method are primarily economic.

A little over a year ago I encountered a non-inguinal hernia which occurred after a clear cut history of trauma. A man about 50, a bank cashier, stooped to lift a ledger from the safe to a high desk, and as he did so he was distinctly aware of something ripping in his back. He had no pain, he said. He thought it was his vest or shirt ripping and let it go at that. In about three weeks, he began to have a little discomfort in his back. This grew a little more intense, and after a month he went into contortions before a mirror, trying to look at his side. Finally he perceived a slight bulge in the lumbar regon. The pain continued and nine months later I saw him. There was a definite and slightly tender bulge when he would bend over and produce tension on the side. At operation I found a protrusion of fat which could easily be reduced, but would reappear on straining. There was a

small tear, perhaps ¾ inch long, in the fascia, in the upper triangle of the lumbar space. We repaired it, and he had no further trouble.

This case, I feel, was of traumatic origin, and it is the only case of upper triangle lumbar hernia that I ever saw.

RALPH G. CAROTHERS (Cincinnati): I think we all agree that Dr. Moorhead has presented a brilliant paper; we all agree there are a great many hernias and that they are a surgical problem. However, the the discussion has brought out the fact that they are brought out social rather than industrial problems. What difference does it make in the long run whether industry pays for the operation, or whether the government takes the money from industry and operates on the fellow through state funds?

We must get these thousands of people operated upon, or injected, or trussed, or out of the way somehow or other, and Washington is going to see that the country pays for it in one manner or another.

HOMER D. DUDLEY (Seattle): Dr. Moorhead has not made a single controversial statement from a purely academic point of view, but how we may apply our services to the public is another matter.

It has been my privilege to examine every applicant for employment with a large company over a period of eighteen years, particularly to determine whether or not there was any likelihood of the applicant's developing a hernia. All of the employees, about 400, have been examined every one to two years, and of that group two have developed hernia from alleged physical effort of a competent cause. Both were operated upon and there were findings to indicate each had a congenital predisposition to the development of hernia.

In the State of Washington it makes no difference what we surgeons believe. The Supreme Court and the Workmen's Industrial Compensation Committee have decided that a workman with a hernia who says he didn't have it prior to any given act or physical effort has a hernia in the eye of the law and comes under the provisions of the Medical Aid Act.

We also have an affidavit form referred to by Dr. Kreuscher which would seem to indicate that there could be excluded the group particularly predisposed to hernia or patients who might have had a hernia. However, all get operated upon and are awarded benefits just the same. The affidavit has been a waste of time and effort, and I believe it has been abandoned.

I have testified in court in regard to all of Dr. Moorhead's criteria, but, unfortunately, no credit is given them by juries. The jury decides that if a man did not have a hernia before the alleged accident and had one afterward, the injury or accident must have been the cause.

Possibly, if an acceptable form could be developed whereby some evaluation could be made between the predisposing factors in the individual

and the disability attributable to injury, there might be some method of presenting this to the courts so that an award could be made only for the percentage of disability due to the injury.

JOHN J. MOORHEAD (closing): This does not seem so much like a closing as it does an opening. It looks as if we had started something.

The first sentence which I used in this paper was to the effect that this was a controversial and perplexing question. That seemed to be borne out a good deal by the discussion.

I may say that in so far as the Compensation Board in New York is concerned, it does not make very much difference what our preconceived notions are, or what the anatomic basis may be, or what the surgical or pathologic findings may be. Their viewpoint is—the man didn't have it before he got hurt; he has it now—and that is the end of that.

It seems to me if an organization like this one could put itself on record from a clinical standpoint, based on the experience of such a large group as this in respect to some of these controversial problems, we would render a real service.

Is there such a thing as a traumatic hernia? Is there such a thing as traumatic appendicitis? Is there such a thing as a traumatic goiter, or traumatic tuberculosis, or traumatic malignant disease? Do we see it? And if this group does not see it, where is it? It seems to me that is one thing that eventually this organization could give itself over to and do a very useful piece of work.

In regard to the bed period for these patients, I remember some years ago we used to keep our cases of appendicitis in bed for four weeks. It got down to two weeks, then ten days, and some people get them up in twenty-four hours. I have gone through exactly the same experience in regard to my hernia cases. I do not see the use of keeping a patient in bed for a period of three or four weeks. My patients develop a disuse of the muscles of the abdominal wall if I keep them inactive too long. My experience is that I get fewer recurrences when I apply the principles that I have just narrated than I did hitherto.

The same applies to the transplantation of the cord. I dislike interfering with the deeper structures, as varicocele or enlargement of the testicle may result, which defeats one of the objects of operation. I cut down the size of the cord sometimes if it has a lipoma or too many veins, but ordinarily I believe it is the result of a failure of circulation, that if I can restore the parts and get the sac out, then that will spontaneously take care of itself.

In relation to white collar labor by comparison with hard workers, the proportion is not so great as you might think. I have a great many patients who are employed in banks in New York City, and the ratio of hernias in that white collar group to the hard-working class is very sur-

prising. I think the thing basically comes down to the anatomic type of the individual.

That goes for the kind of employee to reject. We have bases of preëmployment requirements, but I must say that I am amazed to see how great a number who come in with dilated rings and potential hernias have gone on with hard jobs and yet have had over the years very little in the way of aggravation.

I use no support after operation; I do not use this in any form of laparotomy.

As to proof of aggravation caused by a particular accident, I lay particular emphasis on the question of pain. This is very largely a social as well as a surgical problem.

I do not believe that hernia is a universal bar to employment; as I have already indicated, I know a great many men who are doing laborious work with hernias on their way, who are carrying on their jobs quite effectively.

TRAUMATIC RUPTURE OF URETHRA

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"WE are dealing with a very grave and desperately urgent accident, where simplicity of technique is of paramount importance," said Hamilton Bailey of urethral rupture. Rupture of the urethra may be due to external causes, such as gunshot wounds, stab wounds, blows, falls or crushing injuries, with or without fracture of the pelvis. Vincent O'Conor, reported in 1936 eight cases of rupture of the urethra due to falls astride manhole covers. A careful review of the literature failed to reveal other reports of this type of accident as the cause of urethral traumatism. The blow received by the perineum in these cases is severe because the metal cover is swung violently upward and strikes as the body is lurching downward and forward.

Internal injuries causing urethral rupture may be the passing of instruments, the presence of a foreign body or large rough calculi.

Partial or complete ruptures are classified anatomically as: (1) pendulous, (2) bulbous—anterior to the trigonal ligament; (3) Bulbomembranous—anterior trigonal ligament; (4) membranous portion posterior to the triangular ligament.

Extravasation follows the fascia planes. In lesions anterior to the triangular ligament it spreads over a route limited by Colles' fascia in the scrotum, perineum and penis and in the abdomen by Scarpa's fascia. If the lesion is posterior to the triangular ligament the extravasation involves the retroprostatic region and invades the upper inner aspect of the thigh, the ischiorectal fossa and the buttocks. If rupture is between the layers of the triangular ligament, extravasation may spread externally or toward the pelvis. When intrapelvic rupture is present, it extends to the prevesical and perivesical region.

CASE REPORT

On July 16, 1936, Dr. H. A. R., age 63, while visiting at the home of a colleague, stepped backward out of a car upon a loose cover plate of a manhole which turned. His left leg went down into the manhole, and the perineum struck the edge of the manhole cover.

Examination at Columbia Hospital, one hour later, revealed marked infiltration of blood in the perineum extending completely around the anus and over the inner side of each thigh and into the dorsal surface of scrotum, with bleeding from the meatus. The pulse was weak thready, 120 per minute. Rectal examination revealed a large hematoma the size of an orange extending from the perineum to the apex of the prostate, the prostate being freely movable. A diagnosis of traumatic rupture of the urethra, accompanied by hemorrhage and shock was made.

The patient was immediately prepared for suprapubic and perineal operations. The first operation was done in lithotomy position with a filiform in the urethra. An inverted U incision was made in the perineum through the skin and superficial fascia, which exposed a large blood clot with continuous bleeding from the wound and meatus, when the clot, which had an odor of urine, was removed. The recto-urethral muscle was found to be fragmented, the finger was passed along the rami of the ischium on the left, and the filiform was found outside the urethra. The Young technique was carried out by cutting the central tendon. The filiform was then removed from the urethra and a 15 F. sound passed, which showed the urethra to be torn completely off at the apex of the prostate.

The prostatic urethra was located with some difficulty. A 22 F. soft rubber catheter was passed into the bladder, and urine and blood were obtained. The catheter was anchored to the edge of the membranous urethra with chromic 0 catgut. The torn puboprostatic ligament was then united with one chromic 2 catgut mattress suture. The distal end of the catheter was then pushed over the end of the sound and the sound withdrawn. End-to-end anastomosis was carried out, using chromic 0 catgut, all sutures being inserted before any were tied. A small Penrose drain was inserted on each side and the wound closed with through-and-through silkworm gut sutures, with a small drain in the subcutaneous fat.

A second operation, extraperitoneal suprapubic cystotomy, was then done. The bladder was filled with blood clots and a No. 30 mushroom catheter was used for drainage, with one Penrose drain in the prevesical space which contained free blood. Operative time was fifty-five minutes. During the operations 1500 c.c. of 5 per cent glucose in normal saline was given intravenously. The blood pressure rose from 70/50 to 95/55 during the operations.

At 4 P.M., intravenous glucose, 1500 c.c. in normal saline, was started and at 6 P.M. there was marked improvement in the pulse. The blood pressure was then 116/74. At 11 P.M., a blood transfusion of 500 c.c. was given by the Kimpton-Brown tube.

On July 19, 1936 three drains were removed from the perineum and one from the prevesical space. On July 23, the suprapubic tube was removed. On the tenth postoperative day severe bleeding occurred from the left side of the perineal wound, which was controlled by pressure against the perineum. The systolic blood pressure fell from 150 to 102 and the pulse increased from 80 to 120.

On August 1, 1936 all sutures were removed and the catheter was also removed from urethra. Negative suction at 5 pounds pressure was applied to the suprapubic wound. Two days later the patient was voiding every three hours, and in another two days the suprapubic wound was healed. The patient was then voiding every two hours, and was discharged from the hospital.

On August 10, Van Buren sounds 16, 18, and 20 were passed. The patient remained in the care of Dr. Alexander Randall of Philadelphia for one year. He is now in excellent health and has no stricture.

COMMENT

Verguin of Toulon, in 1757, was the first to employ retrograde catheterization. Guyon in 1892, demonstrated the value of end-to-end anastomosis. Rutherford of Glasgow, in 1898, combined suprapubic and per urethram drainage.

Partial rupture has been treated successfully by suprapubic cystotomy with retrograde splinting of the urethra by an indwelling catheter by George G. Davis, O'Conor, Hutchins, Wheeler and others. Owing to the extravasation of blood and urine and the great advantage of end-to-end anastomosis, it is advisable to do a Young perineal section with the suprapubic drainage.

During the past twenty-six years, I have seen ten cases of rupture of the urethra, including two of the pendulous; two of the bulbous; three of the bulbomembranous; and three of the membranous posterior to triangular ligament. The case reported here is the only one of complete tear in my experience.

Indwelling catheters should be used with care, as they frequently produce traumatic urethritis and cystitis, and if they extend too far into the bladder can ulcerate through the bladder wall, producing peritonitis.[3]

SUMMARY

Traumatic rupture of the urethra due to stepping on a loose manhole cover is more common than current textbooks indicate.

Treatment consists of immediate (1) suprapubic cystotomy and retrograde catheterization; (2) perineal section for end-to-end anastomosis of urethra, suture of puboprostatic ligaments (if torn) and adequate drainage; (3) treatment of shock and hemorrhage by intravenous fluids and blood transfusions, etc.

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DISCUSSION

E. T. CROSSAN (Philadelphia): My compliments go to Dr. Carson for the beautiful result of his case.

That Dr. Carson's results are worthy of comment, you will readily recognize if you have had any experience in trying to find the proximal end of a torn urethra. Furthermore, if you expect that a sutured urethra will remain in contact, you are stretching optimism.

Dr. Weil's method is directly opposite to that which has been described by Dr. Carson and again we all would be stretching optimism if we would hope to obtain the same good results as he has.

In the first place Dr. Weil has been very modest in describing how he finds the bladder; you might find it quite difficult to locate the bladder in a case of fracture of the pelvis in which there has been a great deal of extravasation of urine. Occasionally you might find that you had entered the peritoneal cavity instead of the bladder.

The point of these remarks is this: Since Dr. Weil has been able to obtain good results by retrograde catheterization and leaving the tube in place, why is it not feasible to catheterize the bladder with a metal catheter through the penis at the outset. In those cases that are seen early, the catheter may be left in place to drain. Might this not be preferable to the extensive operations described by Dr. Weil, and equally extensive operation that was described by Dr. Carson?

I know that in proposing such a method I lay myself open to many objections, in that I have not provided for the care of infection. However, the catheter which is used to drain the bladder will at all times act as a drain for the area that has been contaminated. All this depends upon success in getting the catheter in. This would seem to be the first procedure to be attempted. In a few cases we have used the method, and so far with good success.

I should like to ask Dr. Weil what he does in that type of pelvis fracture in which there is a ruptured urethra and the depression of the entire central fragments of the pelvis or the entire symphysis and the ramus of the pubis and the ramus of the ischium have descended in toward the perineum.

CARLETON MATHEWSON, JR. (San Francisco): The most important question that confronts us in traumatic rupture of the urethra is that of diagnosis, which has not been stressed by Dr. Carson. We realize that only

a small percentage of patients with fractures of the pelvis have trauma to their urethra or to their bladder, but because the percentage is so small these individuals are very apt to be overlooked.

On the Emergency Service at the San Francisco City and County Hospital a routine procedure is carried out in all patients presenting themselves with fractures of the pelvis. A voided specimen of urine is examined for evidence of gross or microscopic blood. If the patient is unable to void or if there is evidence of blood in the voided specimen, a catheter is passed into the bladder and a sample of urine obtained. After the bladder has been emptied a measured amount of sterile salt solution is instilled into the bladder, allowed to remain for a few minutes and then removed and measured. If there is any discrepancy in the amount placed in the bladder and that recovered, one immediately suspects the possibility of a rupture in the urethra or in the bladder. If a discrepancy exists, a cystogram and urethrogram are performed. Regardless of this routine procedure, occasionally a rupture of the bladder or of the urethra is missed.

One of our patients, a boy, was in an automobile accident, and sustained a fracture of the pelvis with a marked separation of the symphysis. He was unable to void at the time of entry to the hospital. A catheter was passed into his bladder with ease and urine obtained which contained a very small amount of gross blood. Sterile salt solution injected into the bladder was recovered in the same amount as injected. A cystogram outlined what seemed to be a fairly normal bladder. The bladder was then allowed to empty, and as the catheter was withdrawn a small amount of opaque substance was allowed to run into the urethra. At the end of this examination it was decided that the boy did not have a rupture of the bladder or of the urethra. However, very soon thereafter he developed clinical signs of a ruptured urethra, and the cystograms were reviewed with the roentgenologist. It was at this time that it was first noticed that the bladder shadow in the cystogram was exceptionally high in relation to the symphysis pubis and that there was a small indentation in the side of the bladder in the first cystogram which persisted during the emptying of the bladder. The roentgenologist diagnosed a rupture of the posterior urethra external to the sphincter.

The patient was then operated upon and we found a rupture of the posterior urethra with extravasation of blood around the neck of the bladder which accounted for the indentation seen in the cystogram.

KELLOGG SPEED (Chicago): I have handled quite a large number of such cases, one of the most peculiar of which was that occasioned by a patient sitting on a croquet mallet, balancing himself on the cross bar. A twist of the body fractured the handle of the croquet mallet, causing a long spiral point of the wooded handle to penetrate through the perineum into the urethra.

In the fractures of the pelvis during the War and subsequently, I have handled a large number of gunshot injuries of the urethra and pelvis, and

in over 200 closed or open fractures of the pelvis I have seen urethral involvement in not much over 2 per cent.

Stress of the discussion might very well be laid on the optimism with which the essayist treated his case, because in hospital experience in the large cities there is an enormous mortality from rupture of the urethra, due as has been said, to delayed recognition. Patients may lie for three, four, or five days in the hospital bed without definite knowledge that the urethra is torn or ruptured. The high mortality, of course, is caused by the pressure necrosis from the extravasated urine in the perineal tissues in front of the triangular ligament with absorption and a rapid toxemia. Even wide incision and multiple drainage incisions will fail in many such cases to preserve life, and so a very early recognition is necessary. In routine treatment all fractures of the pelvis should be suspected of caresing ruptures of the urethra and perhaps the first thing the patients should be required to do, if conscious, is to attempt to urinate. If urination cannot be obtained naturally in two hours, they certainly should be catheterized to ascertain whether rupture is present and then suitable surgical measures such as have been shown here must be instituted.

The delayed cases carry statistically a mortality of over 50 per cent.

WILLIAM J. CARSON (closing): I want to thank the speakers for their excellent discussion. As the majority of these cases are not complete ruptures, the treatment is more simplified.

As Hamilton Bailey pointed out, the more simple the treatment the better. I have used the method shown by Dr. Weil and on several occasions, over long distance telephone, have advised surgeons to do the suprapubic cystotomy first and pass a catheter through the prostatic urethra into the torn part of the perineum; second, to make an incision in the perineum over a sound, withdraw the tip of the bladder catheter out of wound, remove the sound, pass a catheter through the urethra and tie the catheters together with heavy silk, then withdraw the suprapubic catheter until the urethral catheter extends into the bladder 2 cm. That will suffice in many cases, but if there is a complete tear and only that splinting is done, a stricture will result and last throughout life.

Extravasation of urine is the most dangerous complication and I feel that Hamilton Bailey has pointed out its important aspect. "You cut your mortality in half by draining through the perineum."

TRANSIENT FALSE MENINGOCELE—
A CLINICAL ENTITY*

PRELIMINARY REPORT

EUGENE J. BOZSAN, M.D., F.A.C.S. AND

THOMAS I. BRENNAN, M.D., F.A.C.S.

NEW YORK CITY

THE escape of cerebrospinal fluid under the scalp through a traumatic opening in the skull is known in particular since the publications of Billroth and Marjolin. Since then, this occur-

FIG. 1. Large collection covered by normal skin.
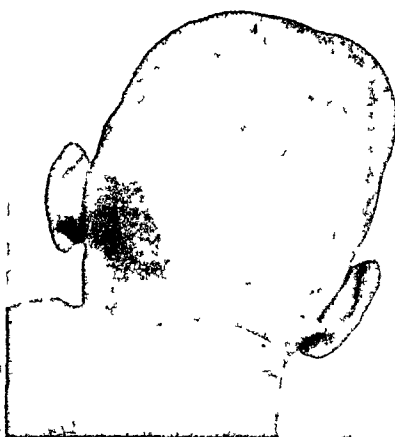
FIG. 2. Small collection covered by normal skin.

rence was observed in a number of instances and a survey of literature made by the authors has yielded over eighty publications bearing directly on this phenomenon, which has been described predominantly under the name of "false meningocele" as contrasted with congenital true meningocele.

The condition was considered a more or less stationary one and tractable only by operative procedures or repeated aspirations, often followed by fatal meningitis. Its occurrence was believed to be rare, and few authors reported more than a single case or at best a small number of cases of their own.

Observation of a large clinical material of head injuries during the last ten years, however, has convinced the authors that a particular type of false meningocele bearing constant features, occurs rather frequently in children following blunt head injuries.

FIG. 3. Hematoma showing discoloration

FIG. 4. Hematoma showing discoloration and bruise.

The main characteristic of this type of cerebrospinal fluid collection is that it disappears spontaneously without any treatment and that in its presence a favorable outcome may be predicted even in the face of rather severe status immediately after the accident.

The authors have observed during the last ten years twenty-nine cases. They believe, however, that this escape of spinal fluid occurs more frequently and that cases are overlooked or are erroneously registered as "hematoma of the scalp." This conjecture is corroborated by the survey of literature since papers dealing with the well known benignity of skull fractures in children often report the presence of hematomata in the case histories.

The authors believe that these so-called hematomata themselves are the reason of the relative benignity of the observed cases, as they contain not blood or not only blood, but spinal fluid, and that through this temporary subcutaneous reservoir, the cranial cavity efficiently decompresses itself.

The clinical features of this type of false meningocele which, because it disappears spontaneously, the authors have come to name transient, are as follows:

Immediately or days after a blunt head injury, a small or large swelling appears on the head, covering at times the whole of the cranial vault. The swelling is painless. Children permit the palpation
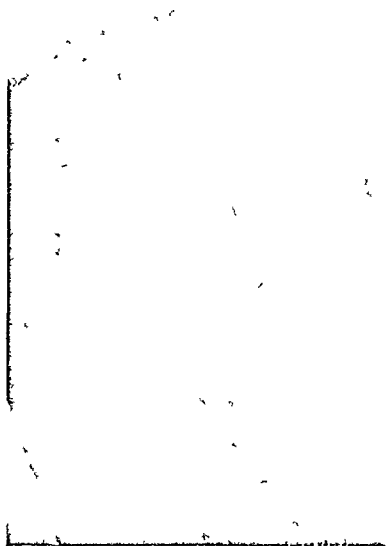
FIG. 5. Typical x-ray picture of false meningocele.

FIG. 6. Typical x-ray picture of hematoma.

of it without fear. The covering skin shows no trace whatever of the irritating presence of extravasated blood, edema, bruise or ecchymoses. During its total stay, no discoloration occurs either early or late, blue or yellow. In most csase the swelling shows loose watery fluctuation, like that of a partly filled hot water bag, often permits palpation of the underlying bone and never discloses snowball crepitus. It transilluminates with the pink hue of a hydrocele. A day or so after its appearance, a circular rim appears at the base of it, which leads to the erroneous conclusion of depressed fracture and is often described by the interns as a "crater."

This clinical picture is strikingly different from that of a true hematoma which is painful, surrounded by edematous skin, shows crunching crepitus of coagulated blood, exhibits bruises and ecchymoses and soon becomes discolored, first black-blue and later yellow. Figures 1 to 4 show clearly the different appearance of the two.

This difference is also apparent by x-ray. The films taken with soft part technique show that the skin over the false meningocele (Fig. 5) is of uniform thickness over the whole extent of the swelling and it is the same as that of the surrounding skin. Under the skin and

separated from it by a narrow cleft is a smoothly outlined shadow of even density hugging the surface of the skull. All structures, bone, fluid and skin, are clearly separated and their borderlines are not blurred or lost.

FIG. 7. False meningocele appearing five days after accident.

FIG. 8. Same as Figure 7, with total disappearance of collection in seven days.

The x-ray picture of a hematoma shows (Fig. 6) in contrast to the above, the skin increasing in thickness from the periphery to the apex of the swelling, spotted with blotches of bloody infiltration and the main mass of hematoma fused with the edematous skin.

Aside from this different clinical appearance, the presence of spinal fluid is indicated by the at times rapid disappearance of the swelling without leaving any trace, and not followed by thickening or fixation of the scalp.

In contrast to this, the hematoma lingers long and fixes the scalp for a considerable time.

No hematoma disappears so quickly and completely as the False Meningoceles illustrated. (Figs. 7–10.)

Against hematoma speaks further the belated appearance of the swelling, at times many days to two weeks after the accident, generally after resumption of more vigorous activity. In one of the cases, the appearance of the collection two days after the accident was preceded by a period of restlessness and drowsiness, indicating increased intracranial pressure which simultaneously disappeared
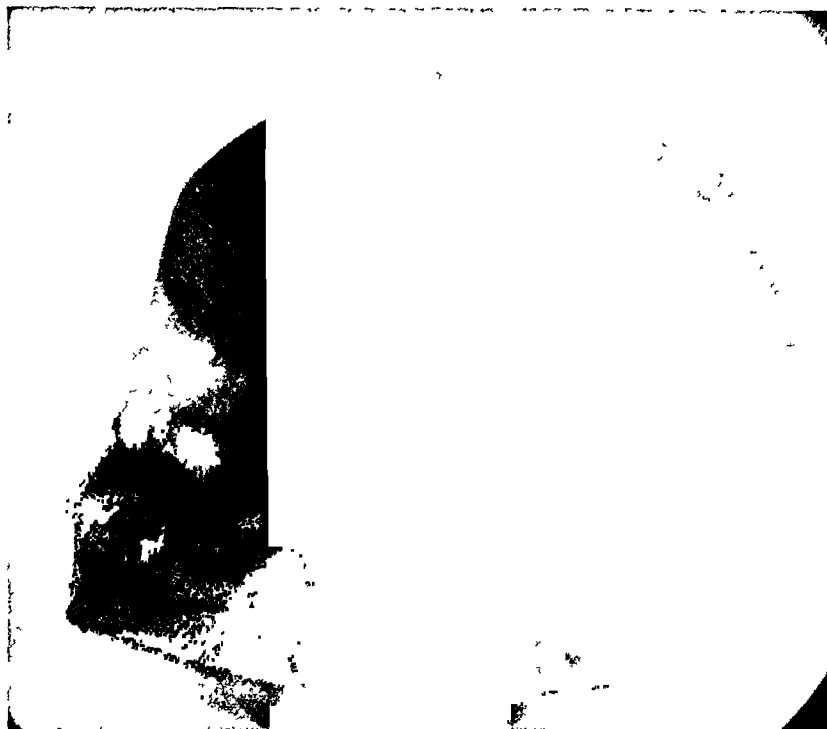
FIG. 9. Complete false meningocele. (August 29, 1930.)
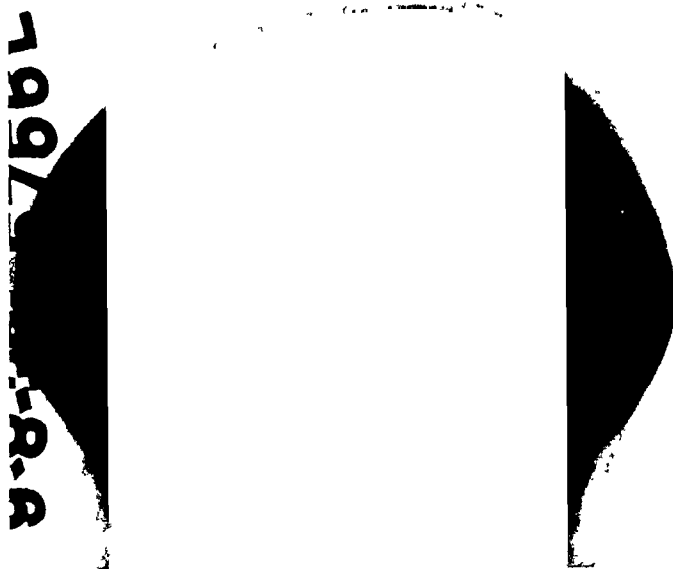
FIG. 10. Same case as Figure 9, showing total disappearance of
collection after ten days.

FIG. 11. Typical vertical fracture line.

FIG. 12. Typical horizontal fracture line.

with the appearance of the swelling. In another case, spinal puncture made the swelling disappear, to reappear the next morning.

Circumstantial evidence, as mentioned above, seems to prove
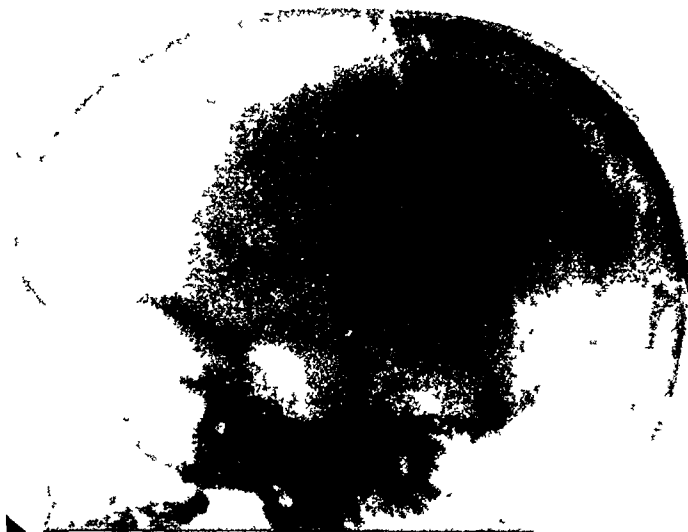
FIG. 13. Typical symmetrical fracture line over both parietal bones

that these swellings contain mainly spinal fluid even if mixed with blood. The authors are definitely opposed to the practice of *proving* the presence of spinal fluid directly by puncture and aspiration. The literature records too great a number of fatalities after aspirations to make this procedure safely practicable.

Puncture and aspiration, however, were performed in our group of cases four times, always on the basis of erroneous diagnosis of hematoma, and in these cases the presence of spinal fluid was proved. In one case, by two repeated aspirations, a total of 235 c.c. of "bloody serum" was removed, the collection returning after each aspiration to disappear spontaneously later. In two other cases, aspiration also proved the presence of spinal fluid. In the fourth case, aspiration was performed only after an "influenza bacillus-like" organism had caused meningitis. In this case, aspiration produced milky pus without any trace of blood. The autopsy of this the only mortality in the group, showed the communication of the cranial cavity with the collection under the scalp.

Beside the constant clinical features, the mechanism by which this escape of spinal fluid under the scalp is brought about is also identical. It accompanies that type of skull fracture in children called indirect, that is, where the fracture occurs not at the site of the

FIG. 14. Typical complete collection.
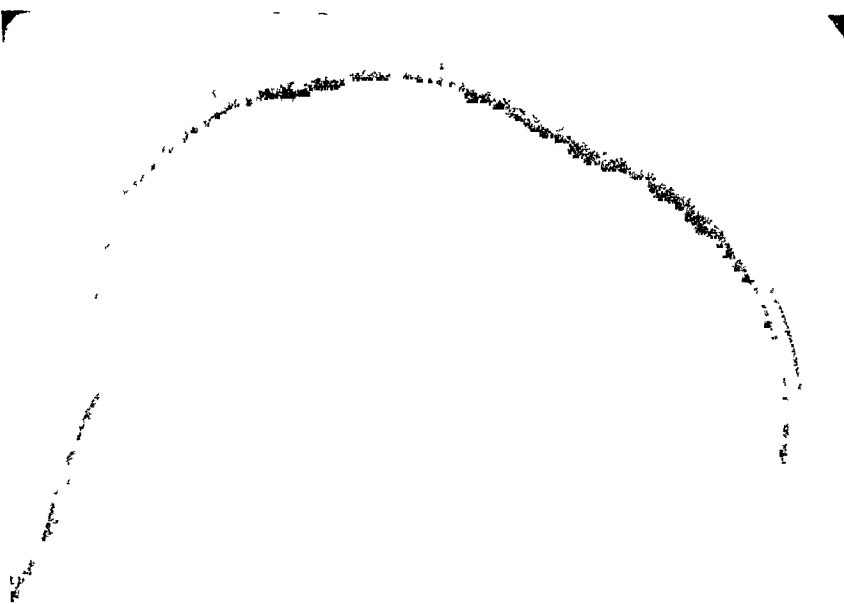
FIG. 15. Typical complete collection.

attack of the force, but at a distant place. These fractures are some-
times called fractures by "contrecoup." There seems to be a differ-
ence in the use of this latter term among different writers, some using
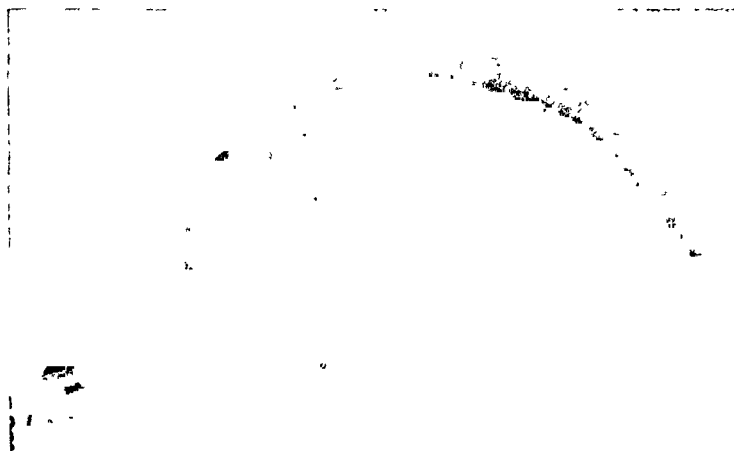
FIG. 16. Partial collection.

FIG. 17. Partial collection.

it to designate injuries to the brain tissue opposite the point of
attack of the force, through hurtling the brain by its momentum or
semi-fluid wave against the inner side of the skull at this point. For
this reason the term of indirect fracture is chosen.

The mechanism of these fractures is well understood and simple.
The elastic skull of a child, compressed by the force of the impact

undergoes a momentary alteration of shape, the maximum of which is located somewhere at the equatorial plane in relation to the point of impact. At this plane of maximum bending, if the force is sufficient,

FIG. 18. Smallest collection observed.

a linear fracture will occur. If the infantile skull is compressed in the fronto-occipital direction, this fracture occurs in a line running over the top of the skull in the frontal plane. (Fig. 11.) If the compression occurs from side to side, the fracture line runs in fronto-occipital direction, higher or lower. (Fig. 12.) In the latter case, the fracture line is most of the time continuous, in the former it may be present only over the maximum convexity of the two parietal bones and is interrupted in the middle line. (Fig. 13.) Of course the skull may be compressed in any meridian, the above-mentioned rule remaining still demonstrable but due to various factors, not as manifestly.

It is these indirect linear fractures over which the transient accumulations of spinal fluid occur.

In the child, the bones of the skull lack diploe and are intimately united with the dura. If fracture occurs, the dura will tear. If intracranial pressure increases due to any reason, this may burst the arachnoid and spinal fluid escapes. If the arachnoid remains intact or the fracture is incomplete, the spinal fluid will not escape immediately after the accident, only some time later when the fissure line becomes widened by absorption or the patient assumes more vigorous activities than during the days immediately following the accident.

In these latter cases, the swelling occurs after a shorter or longer period following the accident.

These accumulations of spinal fluid may cover the whole skull.
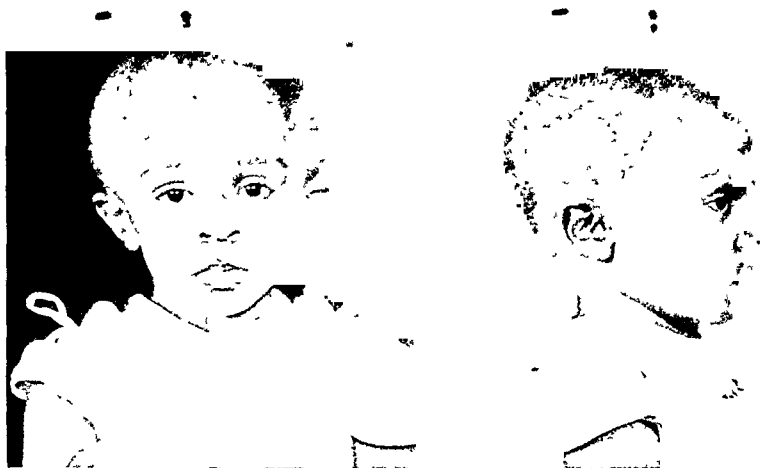
FIG. 19. Symmetrical appearance of a double collection.

Figures 9, 14 and 15 show three of the four complete cases in this series. Most of the time the collections are smaller. (Figs. 16 and 17.) In Figure 18 the smallest collection observed is illustrated.

In cases of symmetrically located fractures the fluid accumulations will assume a strikingly symmetrical aspect. (Fig. 19.)

These indirect fracture lines seldom run into the base. The elastic alteration of the shape of the vault absorbs most of the force. Occasionally, however, as compression exerts its force over the whole bony sphere of the skull, the fracture line extends to the base or a separate fracture line may correspondingly exist there. From these, some slight bloody infiltration originates. As signs of these supposedly bloody infiltrations, we have observed marked pallor of the face as compared with the hands, indicating irritation of the sympathetic nerves. Ecchymosis of the eyelids, passing palsy of the eye muscles, and double vision, were occasionally observed. In one instance a group of herpes vesicles appeared along the course of the infraorbital branch of the fifth nerve, interpreted as a sign of irritation of this nerve at the base.

The authors have assumed as a justified conclusion that in these accumulations, a constant absorption of spinal fluid takes place, much like the accumulation operatively created in the treatment of hydrocephalus. This in their opinion establishes an efficient decompression, not only by forming a temporary reservoir, but also by

eliminating a quantity of the excess fluid. How fast this absorption is remains a matter of conjecture. Concluding from the at times surprising rapidity of disappearance of these accumulations, the impression of the authors is that the absorption is intensive.

The absorption of the fluid is rapidly followed by the disappearance of the rim. The skull becomes smooth and in two or three days the scalp is again as movable as over the rest of the head. This rim is believed to be new bone and is noticed only around partial collections. In the complete collections this rim is not observed.

With such a large series of cases proceeding to spontaneous absorption without exception, the question arose why so many cases reported in the literature remained permanent. A careful analysis of this latter group led the authors to the opinion that it is the type of trauma that determines the permanent or transient feature.

If false meningocele appears over a gunshot or operative wound or over comminuted fractures due to direct trauma, it tends to be permanent. Due to loss of substance or displacement of fragments, the chances of spontaneous closure are unfavorable. The indirect fractures in children, on the other hand, tend to close as the skull regains its shape after the trauma and they give rise to the transient type just described. To this rule there is an exception, namely, when the mechanism of indirect fractures results in the bursting of a suture line.

SUMMARY AND CONCLUSIONS

The material underlying this report, of which detailed analysis will be given in a later publication, reveals that: The age limit of occurrence of transient spurious meningocele was between 1 and 13 years. One case was observed at the age of 17 and another at 18.

The collection appeared immediately after the accident in seventeen cases, and was belated in twelve cases, one to fourteen days after the accident.

The duration of the collections varied between two and twenty-eight days.

X-ray evidence of underlying fracture was present in twenty-seven cases, absent in two cases.

General condition after the accident was severe in five cases, moderate in ten and mild in fourteen cases.

In four cases, the collection covered the whole vault, in twenty-five cases it was partial.

In two cases the collection was double, symmetrically placed, twenty-seven cases the collections were single.

The authors believe that these passing collections of spinal fluid under the scalp deserve careful attention, and their presence may be considered a favorable circumstance as far as immediate prognosis is concerned.

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DISCUSSION

FRANK H. MAYFIELD (Cincinnati): I would like to congratulate Dr. Bozsan and his co-author on the amount of material they have, and also upon the interesting way that they have presented it. I am in accord with the idea that this is probably a clinical entity. It has a counterpart in Dandy's subdural hydroma which, I think, differs from this lesion only in that it connects with the subdural space through a fracture line. I am in accord with the idea that extravasation of cerebrospinal fluid under the scalp is often a protective decompressing mechanism and acts for the betterment of the patient rather than to his detriment.

Dr. Bozsan's criteria for differentiating between hematoma and hydroma are excellent. I agree that it is unwise to aspirate a cavity, or to aspirate repeatedly cavities which communicate with the subarachnoid space.

However, I consider the name badly chosen, for there is in no sense a herniation of the meninges. It is merely an extravasation of fluid underneath the scalp, and I think it would be better named subgalial hydroma or perhaps extracranial hydroma in deference to Dandy's subdural hydroma.

The delayed appearance of the fluid under the scalp which Dr. Bozsan feels is due to rupture of the arachnoid membrane, I would be inclined to explain on the basis that a clot which has been blocking the fracture line had been absorbed and had permitted the extravasation of fluid. At least, I believe, that is the generally accepted opinion as to what happens when leaks appear through the nose or ear later on.

He referred to the rim which could be palpated about the margin of the swelling as possibly being proliferation of bone. This thing appears within a very few hours after the onset of the swelling, and I think it far more likely that it is either a marginal hematoma about the swelling or a false impression of depression, in that the scalp is thinner, stretching from the fluid under it, rather than a proliferation of bone.

While its frequent spontaneous disappearance would certainly indicate that aspiration of the fluid is rarely if ever necessary, I certainly would not have any great fear of infection from the insertion of a sterile needle through a clean area of scalp. I can see no reason why infection should be

more prevalent in this situation than in any ordinary spinal puncture. I can recall two instances in which I thought aspiration was rather helpful.

A child developed extravasation of subdural fluid after injury, and at the same time had a terrific head injury with bloody spinal fluid. He developed signs of irritative meningitis, which so frequently appears in subarachnoid hemorrhage, and we were able to alleviate it by aspirating the cavity through the scalp.

Another patient came in with a small extravasation of fluid similar to those shown, who also had an infected abrasion on the scalp in the neighborhood. The skin immediately over the swelling was intact and clean, and I aspirated it and applied a pressure bandage to discourage recurrence of the collection. I felt happier about it, because I thought we had reduced the chance of getting an infection into the subgalial collection of fluid. You may disagree with that.

EUGENE J. BOZSAN (closing): The name of the condition I did not choose, and I cannot assume responsibility for it. In going through the literature I found this name used in about eighty or ninety publications. I have adhered to it, not wanting to create a new one.

As far as the delayed occurrence is concerned, I perfectly agree with Dr. Mayfield in attributing importance to the absorption of a clot or widening of the fissure by absorption. I have not reported the statistics, in which it is clear that these cases of delayed appearance constitute a large percentage. This fact I believe is also a factor in the opinion that these collections contain spinal fluid and not blood.

About the rim, I am perfectly willing to retreat. convinced though I am that it is newly formed bone, I have not actually seen any; in the one case autopsied I do not recall the report of the examination of the rim. I assumed the formation of the bone in the rim on the analogy with the rim formation in kephalhematomata.

I am opposed to diagnostic aspiration merely because I do not wish to add needlessly even the remote chance of infection.

I omitted to mention one feature. In one of the cases spinal puncture made the swelling disappear, but it reappeared next morning. The very capable resident fortunately made accurate notes of the occurrence.

BRAIN ABSCESS OF TRAUMATIC ORIGIN

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TRAUMATIC abscesses of the brain result from introduction of the infecting organisms by injury, as opposed to the majority of instances in which the abscess is secondary to otitic infections, infections of the accessory nasal sinuses, true osteomyelitis and other diseases of the skull and those of metastatic origin.

In one group of traumatic abscesses, the infection is directly implanted into the brain substance by accidental perforation of the skull with sharp pointed objects, e.g., scissors, knife blades, nails, files, icepicks, etc. The external wound may heal and the accident be considered trivial until an abscess begins to develop, or the external wound may become infected, fail to heal, and the abscess may discharge pus from the wound of entrance. We have seen, on the other hand, two cases of perforation of the brain to a depth of 2 inches through the roof of the orbit by pointed objects without abscess formation or any other apparent permanent damage. The abscess may be secondary to compound comminuted fractures of the skull, usually involving the cranial vault. Meningitis is more likely to occur after compound linear fractures of the base of the skull.

Fragments or spicules of bone, pieces of metal, concrete, hair and dirt, carrying infected material with them, may be driven into the brain substance and an abscess may form around them or in the area from which they were removed. In like manner an abscess may be caused by gunshot wounds. These abscesses usually are completely formed within three to five weeks, are diagnosed and treated with greater ease, and are quite amenable to surgical interference.

In another group, the trauma may be connected more indirectly with abscess formation. Simple linear or comminuted depressed fractures may involve the mastoid, or the accessory nasal sinuses, which already harbor infection. In some instances, an infection of these bony cavities may be induced by the fracture line or lines invading them. In either case, the infection may spread to the brain by any of the known routes associated with the formation of a brain abscess secondary to otitis, mastoiditis, ethmoiditis, frontal sinusitis, etc., namely, by direct extension, retrograde thrombosis and along

the perivascular spaces. At the time the fracture occurs, the dura may be punctured or torn by the margin of a bone fragment. In the absence of compounding externally, infection of the bone fragments of the fracture site through the bony cavity may be delayed. Therefore development of an abscess may not become apparent for quite a length of time.

In some injuries without fracture, the scalp may be badly torn or partly avulsed, with dirt and detritus ground into the outer table of the skull, followed by infection and necrosis of a portion of the skull. The infection may spread to the meninges, produce a localized leptomeningitis, and beneath this area a subcortical abscess may develop.

A compound fracture may possibly or actually be operated upon improperly, the wound may heal and the patient's condition remain good for several weeks or months, only to be followed by formation of an abscess caused by infection retained in the wound of the extra-dural soft parts. Abscess may be caused by improper maneuvers at operation for mastoiditis or disease of the paranasal sinuses. A probe may be pushed through a dehiscence in the inner table and adherent meninges and thus implant infection directly through the meninges. An abscess may develop in the brain after a procedure which can only be mentioned for condemnation, i.e., exploring the brain through an infected dura, either with a needle, cannula, searcher or knife. As many abscesses may form as there are punctures. At a recent autopsy after removal and fixation and section of the brain, three abscesses were seen in the left temporal lobe following three punctures with a needle done by an otologist through infected dura which had been exposed by removal of the dural plate at a previous operation. Unless the abscess is absolutely localized by ventriculo-grams—and not on suspicion—exploratory puncture through in-fected dura is *most strongly condemned*. The lesion producing the ventricular distortion, even in the presence of extradural infection, might be a tumor or cyst. Therefore, in any case, exploratory puncture should be through a clean field. If the dura is carelessly or accidentally torn by a curette during an operation on the mastoid or sinuses, infection may be implanted in the brain substance, but meningeal infection is more likely to ensue.

Foreign bodies—bullets, shell fragments, pieces of helmet, broken-off knife blades, etc.—may be retained in the brain for weeks, months or years without producing undue symptoms. As a result of a blow on the head, or some other cause, the latent infection may flare up and an abscess may develop about the foreign body.

An abscess is likely to follow removal of a foreign body which has been in situ for a long time. In a fairly recent case, a piece of knife blade was removed from the right fronto-parietal region of the brain of a negro man. It had been in the brain for over twenty-eight years and was located by radiographs. He complained only of some weakness of his left hand, for which he was admitted to the hospital. He had no knowledge of being stuck in the head but remembered being "hit on the head and knocked out in a fight over twenty-eight years ago." The knife blade was removed by one of my associates. No evidence of infection existed. A few months later a large abscess, which developed at the site of removal of the blade, was operated on by the author. In his experience, this was the longest interval between the introduction of a foreign body and the formation of an abscess.

Lastly, the brain may be damaged by contrecoup, or a hematoma may be present, and this area may be infected through the blood stream and form an abscess, as in any other part of the body. There may be other types of trauma which serve as a causative factor in the production of a brain abscess, but the more common conditions have been enumerated.

Bacteriology. Where the infection has been carried directly into the brain, the *Staphylococcus aureus* is most often found in the smear and culture. However, any or mixed pus-producing organisms, may be found.

Pathology. Traumatic abscesses when caused by direct implantation of the infection may be small or large. They may be fairly superficial, shaped like a shallow salt cellar or about 2 or 2½ inches deep and elongated like an old-fashioned moneybag. They may also be irregular in shape. They are usually single; some observers consider this to be the case in about 90 per cent of the instances. In the present series, all were single, although one large abscess showed two extensions or pockets on inspection, one rather large, and the other small.

The abscess may become well-formed and well encapsulated early, i.e., within three weeks. A very firm, fibrous wall is usually present, especially near the periphery, due to the presence of a large amount of connective tissue derived from the dura and other tissues. It may, or may not discharge pus through the wound of entrance. The pus may have a very foul, putrid odor, or there may be but little odor. It is thinner and more watery in consistency, also paler than that contained in a nondischarging abscess. In one instance, gas

bubbled from the tract and appeared on the x-ray film to be at the top of the abscess.

In drainage tract abscesses caused by gunshot wounds, and where a tube has been in position for a considerable length of time, a wall about ¼ inch thick in most of its extent forms about the tube. After removal of the tube and healing of the external wound, the partially collapsed cavity may remain quiescent for quite a while. The latent infection may be relighted by a blow on the head or some other cause, and the abscess may suddenly rupture into the lateral ventricle.

Such an instance was observed in the army where a soldier sustained a gunshot wound of the right frontal lobe. An abscess developed in the tract and was drained for several weeks by a rubber tube. The tract ran obliquely backward and outward from a point near the middle of the forehead for a distance of about 3 inches. After removal of the tube, the wound healed kindly and the patient made what was considered a good recovery. He was quite intelligent, had a good working knowledge of airplanes, and demonstrated the mechanics of airplane repairs and construction to other patients in the occupational therapy department.

One night, several months after the healing of the wound and his return to work as instructor, he was in a public house, drank a bit of wine and became somewhat unruly and boisterous. An M.P. was called and, in the attempt to quiet the man, tapped him lightly on the head with a billy. This light blow served the purpose of subduing the man, but did not render him unconscious. He was escorted to his barracks, went to sleep, had a good night's rest and, for the time being, was none the worse for the spree. The following morning at about 10 o'clock, he was sitting at a table playing pinochle with his mates, when he made a sudden outcry, threw up his hands, fell back unconscious on the bed and never recovered consciousness.

On lumbar puncture, milky cerebrospinal fluid was obtained under marked pressure, loaded with pus cells and organisms. The patient died thirty-six hours after the episode. When the brain was sectioned, the long, thick-walled tract was found to be almost completely collapsed, with a narrow space between the two walls containing thick, inspissated pus, the consistency and color of peanut butter. Further section showed a thin portion of the wall through which a perforation into the ventricle occurred.

Many discharging brain fistulae were observed in old infected gunshot wounds of the head, some discharging pus copiously for many months. After removal of a small piece of dead bone fragment with a fenestrated curette, and institution of proper treatment, the fistulae usually closed.

When the abscess is secondary to traumatic involvement and infection of the mastoid and accessory nasal sinuses, it has the

characteristics of brain abscesses complicating primary disease of these structures. In shape they are usually ovoid or spherical. The various stages are edema, softening of the brain, suppurative encephalitis with necrosis, free pus formation and encapsulation. The time required is from three to five weeks after the initial spread of infection to the brain substance. The wall or "capsule" is from 2 to 4 mm. in thickness.

Signs, Symptoms and Diagnosis. If there is a discharging sinus, the diagnosis is fairly evident. If a small tube is introduced into the tract and a large amount of pus is discharged through the tube, the diagnosis is well established. X-ray films may show the cavity partly filled with gas and a fluid level. These patients are not very ill, once the intermittent or continuous discharge of pus takes place.

On the other hand, if the abscess is intact, it exhibits the same signs and symptoms as abscesses of nontraumatic origin—pain in the head, tenderness on percussion, nausea and vomiting, prostration, loss of appetite and weight, slow cerebration, monoparesis or hemiparesis or hemiplegia if the motor area is involved directly by the edema, motor asphasia (if the area of Broca is directly involved or if the abscess is large and is in the dominant frontal lobe), drowsiness and stupor. The deep reflexes are usually increased, especially on the contralateral side, with decrease of the corresponding abdominal reflexes.

Involvement of the cranial nerves will depend upon the location of the abscess. Fundus changes are likewise similar to those in other abscesses. Papilledema is not observed in the early stages, but may be present later. There is engorgement of the retinal veins and there may be hemorrhages. Visual field defects, if they exist, will vary with the location of the abscess.

There is usually a leucocytosis, the count being between 9,000 and 16,000. The cerebrospinal fluid may show a normal cell count, or it may be increased. Manometric readings show increase in pressure.

The diagnosis, as a rule, is made earlier in traumatic than in nontraumatic cases, since the existing head trauma will more often and more promptly arouse suspicion of the existence of a subcortical lesion. The differential diagnosis between abscess and meningitis, suppurative leptomeningitis, subdural and extradural pus pockets, and edema, should be made if possible. The abscess is also more favorable for localization. Should attempts to make the diagnosis and localization fail, one should resort to verticulography, and not encephalography. Displacement and distortion of the ventricular system will almost always indicate the location of the abscess.

Treatment. Prophylactic measures, in general, deal with prompt removal if possible, of the extracranial or cranial source of infection to which the brain abscess may be secondary. Compound fractures of the skull should be properly and completely operated upon, preferably in the first eight or ten hours. It is remarkable, even in this time, how many cases of head wounds are badly cared for. Scalp lacerations are carelessly sutured tightly over infected material. I maintain that every laceration of the scalp should be closed under good light in the operating room and not haphazardly in a dressing room. Some observers believe that the infection of the brain substance takes place simultaneously with the onset of infection of the mastoid and sinuses. This may be true in some cases, but the delayed riddance of infection in these structures is responsible, without question, for many more, and one must admit that, in some instances the direct cause of the infection spreading to the brain is due in a large measure to *delayed* operation, *careless* operation, or *inadequate* operation for infection of the traumatized mastoid or accessory nasal sinuses.

When an abscess begins to develop, provided it is not metastatic, supportive measures to help maintain the best possible general condition of the patient until walling-off of the process has been accomplished, are of primary importance. This walling-off period is from about two and one-half to four weeks in duration. The increased and sometimes high intracranial pressure should be reduced by means of intravenous sucrose solution, 100 c.c. of 50 per cent solution every four hours or less, frequent saline laxatives and cathartics, enema or colonic irrigations, elevated position of the head, and careful lumbar punctures when indicated.

Lumbar punctures for decompressive effect should not be done when the abscess is in the cerebellar lobe. Ventricular puncture through the anterior horn is preferred. Headaches and pains in the head are almost immediately relieved by tapping the ventricle. Cerebration improves and the patient, who may otherwise have been in deep stupor, usually becomes quite alert. Repeated ventricular punctures through the anterior burr hole may be necessary both before and after operation for cerebellar abscess. The anterior position of the burr hole mitigates against infection from the operative wound after the abscess has been drained, and the site is also convenient for tapping.

Operation should be done after a well formed wall has developed. The period required may be no longer than 2½ weeks. Otherwise, the approved time is from twenty-one to thirty days. In a majority of

cases seen by the neurologic surgeon, the abscess wall has already become well established, and no delay of the operation is necessary. In fact, a few patients may be so ill that the operation may have to be done as an emergency, but this situation is rare due to increased knowledge regarding dehydration and decompressive measures.

Until the patient comes to operation, at the hands of a capable surgeon, experienced with the conditions under consideration, it would be much better if the inquisitive meddling searcher-for-an-abscess could adopt an absolute "hands off" operative policy. If such an attitude existed, one would not so often be called upon to attempt to save the life of an individual whose condition is not unlike that of an inexperienced swimmer from whom one has taken a lifebelt. I have for some time held the opinion that one should not boldly plunge in, smugly relate afterwards that he "struck pus" (a favorite expression), watch one complication after another develop until the situation is well out of hand, and then hurriedly and sometimes patronizingly dump the remnants of a case into the lap of a colleague.

Several procedures, with modifications, have been advised by various writers and observers. The main objectives and desires should be the preservation of the life of the patient, removal of the pus with subsidence of brain edema and healing of the wound with recovery of the patient and return to his normal life.

The aim at operation is the *prevention* of meningitis, rupture into the ventricle, repetition of infection of the brain, multiple operations for the same single lesion, paralysis, convulsions and death. It is very doubtful if any described operative procedure would meet all of these requirements and would result in 100 per cent cures.

In cerebral abscesses I have employed for several years an operation, the chief principles of which were advocated in 1923. On several occasions I have published a description of this method, and it is not necessary to repeat it here. Suffice it to say that an opening is made in the skull, dura, brain cortex and abscess to provide a *direct* approach, entrance and avenue into the abscess cavity. One can get rid of the pus once and for all, enlarge the opening sufficiently to allow thorough inspection of the interior of the abscess cavity, see whether openings into secondary pockets or extensions exist, and, if they do, deal with them in such manner that they become a part of the main cavity and do not remain like a cellar or attic stored with dynamite. After a complete survey has been made, the abscess cavity is slowly effaced, leaving no trace, by controlled gradual elevation of the floor of the abscess *up to*, but not *beyond* the level of

the skull. One might ask what becomes of the cavity, and this question can be answered by stating that it simply *disappears*. It does not remain in the cranial cavity. It forms a part of the scar on the brain surface, granulates over, becomes covered with epithelium, like any scar, and the area heals.

Scalp plastic is usually performed about six months or one year later. The scar is excised and normal scalp edges are brought together.

REPORT OF CASES

In sixty cases of brain abscess which have come under the observation of the author since 1920, only seven were of traumatic origin. Three cases have already been reported more or less in detail, and four more will be reported in this paper.

CASE 1.   *F. R., male, white, 56 years old, a barber, was admitted to the Jersey City Medical Center, February 2, 1935. He had a brain abscess in the left postfrontal region, resulting from the introduction of a penknife into the brain through an operative cranial defect made ten years previously. Operation was followed by recovery.*

The patient was picked up in the street by the hospital ambulance. He was in a comatose condition and had a deep laceration of the scalp. The history was later obtained piecemeal from relatives and from the patient during his hospitalization. Early in the morning on the day of admission he had awakened a member of the family to ask the time. About three hours later the family arose and found that the patient had left. On the bathroom and bedroom rugs there was blood, and about half a cupful in a pan under the bed. He was not heard of until notification of the hospitalization was received. It was later learned from the patient that he had thrust a "penknife" down "to the hilt" into his brain through an old skull defect made by the elevation of a skull fracture ten years previously. During several months prior to this episode, he had had periods of mental disturbance during which he felt that someone was "after him," seeking his life. Associated with this there was a vague history of abdominal pain. At these times the operative site would "itch," and it was while scratching this area that he introduced the penknife. Until the onset of this mental disturbance he was perfectly well and able to work at his trade as a barber. The patient was admitted to the hospital about four hours after he was known to have been at home.

Past History.   An appendectomy had been done in 1918. In 1923, while riding a motorcycle, he sustained a head injury which rendered him unconscious for an undetermined period of time. He was taken to a hospital in Norwich, Connecticut, and was operated on immediately.

Physical Examination.   The patient's temperature was 98, his pulse 90, respirations 20, blood pressure 135/80. He was an elderly appearing man in a comatose condition and had frequent convulsive seizures of the entire

body. There was a depressed bloody area in the left post-frontal region under which there was definite absence of bone.

The patient was comatose, his pupils were equal and reacted sluggishly to light. The fundi were normal, as were other cranials, motor power, and sensory. The reflexes were equal and hyperactive. A left equivocal plantar response was noted.

Laboratory examination showed the urine normal, the white blood count 10,850, and the red count 4,625,000.

On radiographic examination, there was a skull defect about $1\frac{1}{4}$ inches long and about $\frac{1}{8}$ inch wide in the left postfrontal area, the longest diameter parallel to the anteroposterior diameter of the skull. This had the appearance of an old operative defect for compound comminuted fracture of the skull.

Course in Hospital. For the first six days the patient's general condition remained about the same except that the convulsions subsided. He had a definite "psychosis" in that he thought someone was coming to kill him. He was incontinent of urine at times. His temperature ranged from 99 to 100, and the pulse averaged about 72, with a range of 60 to 90. On about the sixth day there was evidence of infection in the scalp wound.

On the eighteenth day of hospitalization the man became quiet with fixed pupils and weak pulse of 50 or 60, and blood pressure 70/50. A scab was removed from the scalp wound, allowing a large quantity of foul pus to escape. The scalp lesion was then widened with scissors and explored. There was no evidence of a scalp abscess, but a sinus tract leading toward the brain substance was discovered. Culture from this pus was reported as streptococcus.

On the twenty-fourth day the pulse leveled off at a high of 70, and thereafter ranged between 55 and 70 until operation on the thirtieth day of hospitalization, at which time there was definite evidence of a brain abscess and no contraindication for delaying operation.

Operation for brain abscess was done March 4, 1935. When the dressing was removed in the operating room, thick yellow foul-smelling pus escaped from the sinus lying in the middle of the scalp wound. A crucial incision was made over the previously described bony defect centering over the discharging sinus. The cortex was exposed and was seen to be markedly fibrous, with considerable edema. It was firm in consistency. The sinus was easily visualized. Its opening was gradually enlarged by incising the tissue about it, which produced a sudden gush of a large amount of pus. An opening about $1\frac{1}{4}$ inches in diameter was made into the cavity, so that when the pus was removed by suction and thoroughly washed out with saline, the cavity could be inspected. It is estimated that this cavity contained 2 to 3 ounces of pus. The lateral walls collapsed with remarkable rapidity following evacuation, so that introduction of proper drainage material was impossible. A spinal puncture was done with removal of 40 to 45 c.c. of clear cerebrospinal fluid, an amount sufficient to cause the cavity of the

abscess to open widely and allow proper inspection. It appeared somewhat like a cylindrical tract about 2½ inches deep and about 1 inch or more in diameter which led directly downward, with possibly slight deviation forward. Drainage of such a cavity presented a difficult problem, since it was believed that this abscess could hardly herniate from the depths to the surface. Therefore, two strips of iodoform gauze were placed down to the bottom with the intention of leaving these in place for about six days and controlling the intraventricular pressure by lumbar punctures, so that the base might become more superficial, while the vertical walls were held apart. Fluffed iodoform gauze was loosely stuffed in the outer wound and a copious dressing was applied. The postoperative condition was good.

Postoperative Course. On the second day after operation the patient was more mentally alert, had no complaints, and the maximum temperature was 99.8. Thirty c.c. of clear cerebrospinal fluid was removed, and the outer dressing changed. Spinal taps were done every two days, 15 to 30 c.c. being removed at each tapping.

On the ninth postoperative day the first complete postoperative dressing was done. All the original iodoform gauze was removed by loosening with hydrogen peroxide. The cavity was clean and the walls were covered with healthy appearing granulation tissue. The walls were firmly resistant to pressure, but the cavity remained about 2¼ to 2½ inches deep. A spinal tap was done to open it for better inspection. A thin rubber perforated bag was inserted and was mildly packed with strips of iodoform gauze and a wet azochloramid gauze dressing applied. The general condition remained good, without vomiting, headache, or elevation of temperature above 100. It was deemed advisable to decrease the number of spinal taps, keep the patient in a semi-sitting position, with catharsis for intraventricular pressure control. The patient continued to improve.

By the eighteenth day the temperature remained essentially normal, and by the thirty-eighth postoperative day the cavity had gradually become smaller, being about ½ inch deep. Temperature, pulse and respiration had remained satisfactory, and the general condition had improved steadily. On the thirty-eighth day he mentioned that someone was "after him" again; he seemed rather discouraged and wanted to go home. He continued to have delusions of persecution. On the forty-fourth day postoperative, the cavity was practically obliterated, and on the 64th day, May 5, 1935, he was discharged. The operative site had healed, but he continued to have delusions of persecution and intermittent moody spells with paranoid trend. He was sent to an institution and is there at the present time.

CASE II. *G. C. Male, white, 7 years old, was admitted to Bellevue Hospital July 8, 1937. He had a brain abscess of the right frontal lobe, resulting from a compound comminuted fracture of the skull. Operation resulted in recovery.*

The patient had sustained a compound comminuted fracture of the right frontal bone on May 12, 1937, two months before this admission, pro-

duced by the sharp edge of a car door handle striking his head. He was unconscious for a short time. The wound was treated at another hospital where it was reported that a piece of bone was removed from the laceration, with debridement of the skin, and tight closure, without drainage. He had remained in this hospital three weeks and four days. It was said the wound healed without infection, and the temperature did not rise above 101. He was discharged to his home, apparently without clinical evidence of further complications. While at home he became irritable and was sent to Seaside Hospital because of "fits of anger." At Seaside he showed personality changes, became increasingly irritable, disinterested in his surroundings, and complained occasionally of right frontal headache which was associated with a slow pulse, as low as 46 on occasions, with slight elevation of temperature, nausea and vomiting two or three days prior to admission to Bellevue Hospital.

Past History. The patient had always stuttered. He had had chickenpox at 4 years, was vaccinated at 1½ years. His health in general was good.

Physical Examination. During the day and a half before operation, the pulse averaged 70 (55–95), the temperature averaged 99.2 (98.6–100), and respirations 20. Weight was 39¾ lbs.

The child was well developed, coöperative and alert, but showed a disinclination to play with other children. There was a well healed scar over the right frontal region just below the hairline, extending down towards the frontal sinus area, beneath which was definite inequality of continuity of bone.

The speech was stuttering, but not aphasic. The right pupil was larger than left and did not react to light so well as the left. A slight right facial weakness appeared peripheral in type. The hearing was suggestively diminished on right. There was definite papilledema of about 1 or 2 diopters, equal on both sides, with engorgement of veins, more marked on the right. Other cranials were normal. There was no discernible loss of motor power. Sensation was intact. Reflexes: left biceps and left knee jerks were greater than the right; the abdominals were present, equal and active; the plantars were flexor, although the left seemed less frankly so. No skull tenderness, and no stiffness of the neck were observed. Coördination was normal.

Laboratory examination showed W.B.C. 9,000, with polys 72 per cent, lymphocytes 18, monocytes 10. The sedimentation rate was 17 in thirty minutes, 21 in sixty minutes. Urine was normal and Wassermann negative.

At operation on July 10, 1937, under avertin, local and nitrous oxide and oxygen anesthesia, the old scar which was just beneath the hairline on the lateral side of the frontal bone, extending down to within ½ inch of the eyebrow, was excised. A crucial incision was made and the scalp flaps were held back with self-retaining retractors, exposing viable loosely attached fragments of bone beneath the scar. These were removed with a blunt periosteal elevator. The frontal sinus was not involved and therefore was not entered. The dura was absent in an area directly beneath the bony

fragments, but intact at the edges of the defect. A nick was made in the intact thickened dura, and with a blunt brain cannula, the abscess wall was encountered about 1½ cm. beneath the surface, more toward the midline than directly beneath the fragments. The bony defect was enlarged with rongeurs to about the size of a silver dollar. The overlying cortex was removed by suction, revealing the abscess wall. Three narrow strips of iodoform gauze were placed over the edges of the dura and bone to wall off the cortical surface and meninges.

The presenting abscess was then aspirated through a cannula, thick yellow pus being obtained. The presenting portion of the abscess wall was removed, and about 40 c.c. of pus evacuated by suction. A spinal tap was done with removal of 35 c.c. of clear fluid. This allowed the partially collapsed abscess cavity to dilate and permitted inspection of the cavity. The walls were about 3 mm. thick, and no extensions or secondary pockets existed. An iodoform gauze "handkerchief" was placed in the cavity and several soft strips of iodoform gauze were packed into this according to the method advocated.

Culture of the pus from the abscess was reported to be aerobic *Staphylococcus aureus*. No anaerobic organisms were found.

Postoperative Course. The first dressing was done on the second day postoperative, when the temperature was 99, the pulse 80. The general condition was excellent. The patient was singing, drawing pictures, and playing with toys all day. This status continued until the fifth day postoperative, at which time during my absence, the child began to show signs of increased intracranial pressure which was not controlled by spinal taps, catharsis and position. Too great herniation of the abscess floor was allowed, and upon my return on the twelfth day postoperative, hernia cerebri about the size of a lemon was present with complete eversion of the abscess cavity and a mechanical ptosis of the right eyelid by pressure.

With the advent of the hernia, the temperature ranged from 100 to 101. The treatment now became the treatment for the hernia cerebri, i.e., spinal taps, position, catharsis, a protective dressing with perforated strips of adhesive and pressure applied as indicated at each dressing and dakinization.

On the twentieth day postoperative the hernia was smaller, but was being irritated by trauma due to the fact that the patient turned to the right, the same side as that of the hernia, to watch visitors at the door. When he was moved to the other side of the room, the irritation quickly subsided.

The last spinal tap required was on the twenty-eighth day after operation. On the thirty-second day perforated adhesive was applied over the now small hernia to assure more equal and constant pressure and to hasten epithelization. On the thirty-sixth day a compression dressing was applied. By the forty-eighth day the hernia had completely subsided and only a dimple remained at the site of the abscess floor. He was up and about six days later and on the sixtieth day arrangements were made for him to

attend school in the hospital while awaiting the time for a scalp plastic. It was not deemed advisable to discharge the child to his home because of conditions there and the possibility of trauma.

On January 14, 1938, seven months postoperative, the scalp plastic was performed. It healed kindly. An aluminum leather helmet was obtained for the patient for the protection of the pulsating defect, and on February 16, 1938 he was discharged to his home. When last seen he was in good health, attending school, and doing his school work in a satisfactory manner.

CASE III. *S. W., male, colored, 52 years old, a laborer, was admitted to the neurological and neurosurgical service of Bellevue Hospital November 3, 1937. He was discharged November 29 and readmitted February 12, 1938. He had a right parietofrontotemporal abscess, traumatic in origin, secondary to a knife blade removal on his first admission. It had been imbedded in the brain for more than twenty-eight years. Operation was done three months and four days after removal of the knife blade and recovery resulted.*

Four or five months prior to admission on November 3, 1937 the patient had the first of a series of left-sided Jacksonian seizures involving the face and arm without loss of consciousness and usually without convulsive movements of the leg. The exact frequency and duration of the attacks could not be ascertained. The patient's family noted lapses of memory and some mental confusion. Four weeks prior to the above time the convulsions became more frequent, and the movements of the left arm and face were almost "constant," although there was loss of consciousness. On his admission to the hospital the symptoms became less pronounced and headache less severe, and he became clearer mentally.

Past History. There was a long history of inadequately treated lues, numerous undetailed minor head injuries. The patient stated that his scalp was slashed with a knife thirty years before in a fight, that he was knocked down and did not know that he was stuck in the head with a knife. Frequent alcoholic bouts figured in the history.

Physical Examination. The patient was well developed and well nourished. His blood pressure was 122/80, temperature 98.6, pulse averaged 70, respirations 18.

The pupils were equal and reacted well, the fundi normal. Left facial weakness was noted, both voluntary and mimetic. The left corneal reflex was diminished, but there was no hemianopsia. Slight motor weakness of the left face, arm and left leg was observed, but this was not pronounced in the leg. Sensory examination showed extreme loss of position sense in the left hand with complete astereognosis and tabetic pseudoathetosis. There was diminution of all forms of sensation over the entire left half of the body. All tendon jerks were present, greater on the left side. The left abdominals were diminished and the plantars both flexor.

The urine and blood cytology were normal, the blood Wassermann doubtful. Spinal fluid pressure was 100 mm. water. The fluid was clear,

with ten monocytes and a total protein of 40. The Pardy was negative
and the Wassermann negative. Colloidal gold 0011100000.

X-ray examination revealed a "saw-toothed" knife blade in the right
parietal bone just above the temporoparietal suture, extending down into
the substance of the brain for about 2½ inches.

Operation for the removal of the knife blade was done November 15,
1937 under local anesthesia. The butt could be felt when the head was
shaved. A 2 inch incision was made over this area. A burr hole was made
and a circle of bone rongeured out, leaving a defect the size of a half dollar.
The knife blade was intact in the piece of bone. The blade was removed from
the dura and brain easily without opening the dura. A small amount of
yellow fluid escaped after removal of the blade. Culture of this fluid was
negative. The dura pulsated. The area was cauterized and closed without
drainage. There was no evidence of pus.

The postoperative course was uneventful. The headache disappeared
and motor power improved rapidly, as did the astereognosis and difference
in reflexes. On discharge November 29 there was no disturbance in sensory
findings, and it was felt that the patient was cured.

He was not heard of again until February 18, 1938 when another
service in Bellevue Hospital asked for a consultation. The patient at this
time was almost moribund and had been brought into the emergency room
in profound stupor with decubitous ulcers over the dependent portions of
the body. No history could be obtained except that his wife stated he had
been "too sick to come to the hospital for a month."

Physical examination revealed a markedly emaciated stuporous colored
man, reacting but slightly to very painful stimuli, with large dirty decubit-
ous ulcers over the sacrum, buttocks, both heels and right scapular area.
The patient was incontinent of urine and feces. Temperature averaged
99.6, pulse 100 to 120, blood pressure 120/90.

Neurologic Examination.   The neck was definitely stiff and the Kernig
positive. Moderate bulging was present at the previous operative site in
the skull. The head was held to the right with eyes fixed to the right side.
The right pupil was larger than left and both pupils reacted sluggishly to
light. The fundi were normal except for engorgement of veins on the right.
There was definite central facial weakness on the left. Other cranials were
normal as could be tested, except for suggestive complete left fifth nerve
paralysis to sensation. Flaccid paralysis of the left arm and paresis of the
left leg were recorded. Abdominal reflexes were present on the right, absent
on the left. The deep reflexes were diminished on the left. The plantars:
flexor on the right, equivocal on the left.

The white blood count was 9500.

Operation for a brain abscess secondary to removal of foreign body,
was done February 19, 1938 at 2:30 A.M. A crucial incision was made over
the previous bony defect. The scar tissue underlying was removed, ex-
posing the bony edges which contained bone wax which had been used

to stop bleeding at the previous operation for removal of the knife blade. The defect was enlarged to assure the removal of all foreign material. The dural scar was excised and a blunt brain cannula inserted. At about 8 mm. the typical resistance of a brain abscess was encountered. The overlying brain cortex was removed with suction, exposing the presenting tip of the abscess, which was typically brownish red in color. The presenting portion of the abscess was removed and the cavity was inspected. About 2½ ounces of greenish yellow thick pus were evacuated from the abscess, with thick inspissated pus along the walls being removed with pledgets of cotton and suction. Two extensions or secondary pockets were connected with the main abscess cavity. These were clearly visible after all pus had been removed, one anterior and one postero-inferior. The more posterior extension was globular in shape, about the size of a hickory nut and communicated with the main cavity through an opening about the size of the end of a cigarette. The anterior one was smaller. It was possible to dilate their respective openings.

The usual "handkerchief" of iodoform gauze was inserted into the larger original cavity, with perforations made in it to correspond to the openings into the secondary pocket. Through these openings, smaller pieces of iodoform gauze were placed in order to provide adequate drainage and to afford opportunity for subsequent treatment with clear visualization. Strips of iodoform gauze were packed snugly inside the "handkerchief" and the usual copious dressing was applied. The patient became alert at the end of the procedure and seemed to be mentally clear, but was very weak.

Culture of the pus from the abscess showed Gram-positive intra- and extracellular diplococci and *Staphylococcus aureus*.

Postoperative Course. A transfusion of 450 c.c. of citrated blood was given on the second day. Dressings were done daily with spinal taps as indicated. By the fifth day postoperative the mental status was normal, the hemiparesis was definitely improved, and the temperature was about normal. The maximum temperature was 101°F., the day on which the initial packing was removed. On the sixth day postoperative the entire initial packing was removed, and at this time the secondary anterior abscess was obliterated and the posterior pocket was becoming much smaller. The communication with the posterior pocket was gradually dilated with forceps and three gauze packings were then inserted to allow the cavity to become still smaller. This took place rapidly.

An uneventful recovery followed. The bedsores rapidly healed. The patient was sitting up in a wheel chair on the twenty-eighth postoperative day. His weight, which at the time of operation was about 100 pounds, rose to 113 pounds. At this time there was only a small dimple in the center of a granulating area about ⅝ inch in diameter. On the sixty-fourth day there remained slight weakness of the left upper extremity, but this did not prevent good use of the hand. Gait was normal, and but slight difference

in reflexes remained. The patient weighed 138 pounds at this time. He continued to improve and on the day of his discharge, June 13, 1938, he weighed 157 pounds. When last seen he was in good health. A scalp plastic, under local anesthesia, is to be done.

CASE IV. *S. M., male, white, 18 years old, was admitted to Jersey City Medical Center August 26, 1937 and remained until September, 5, 1937. He was readmitted September 13, 1937, and discharged November 23, 1937. A brain abscess, traumatic, in the right temporal region had followed a puncture wound in this area by sharp pointed scissors. Operation resulted in recovery.*

The patient was first seen in the emergency room at the Jersey City Medical Center on August 27, 1937 at which time he presented a laceration of the scalp 1½ inches long over the right ear produced by another person throwing a pair of scissors at him. The laceration was dressed and he was sent home. Four days later he returned to the hospital having stayed in bed at home in the interval, because of pain in his head. The lacerated area had become very painful and was associated with some dizziness.

Past history was irrelevant.

Examination revealed a well developed boy, who was "not very cooperative"; his face was flushed, and he appeared acutely ill. There was a localized area of acute tenderness and swelling over the laceration above the right ear. This swelling was fluctuant and was diagnosed as an infected hematoma of the scalp. The Wassermann was negative.

On admission to the hospital the fluctuating area was incised, drained and packed with plain gauze. Temperature was 100, pulse 68, and respiration 22. About 20 c.c. of foul smelling blood was evacuated but was not cultured. A wet dressing was applied. Antitetanic serum was not given. Daily wet aluminum acetate dressings were applied.

On August 28, 1937, two days after admission, there was a foul-smelling discharge on the dressing, and the patient complained of headache and photophobia with pain to slight pressure over the right eyeball. Temperature fluctuated for the first four days between 98.6 and 101, with a pulse between 65 and 88. By the fifth day the temperature was about normal to 99, with pulse of 72. The patient was discharged September 5, 1937 was a healed laceration.

He felt well for two days, but then developed frontal headache. A local medical man was consulted and reopened the laceration, obtaining pus. Two days following this the patient was nauseated, vomited five or six times, and had a general feeling of malaise. He improved during the next few days and was well until September 12, at which time headache returned, accompanied by malaise, nausea, vomiting and mild photophobia. He was readmitted to the hospital September 13, one week following his discharge from the hospital and twenty-two days after his accident.

At this time his temperature was 99.4, pulse 70, respiration 20. He was acutely ill and showed a definite mental change, being irritable, mildly uncoöperative, emotionally unstable and apprehensive. The face was

flushed. The laceration over the right ear was open ½ inch and was draining foul-smelling thick yellow pus. There was a fluctuant area antero-inferiorly to the laceration, which was tender, red and warm. General physical examination was otherwise negative.

The patient was moderately stuporous. His neck was not stiff. The pupils were round, regular and equal, reacting to light and accommodation. Definite photophobia was present, more marked on the right. The fundi were within physiologic limits with haziness of nasal margins of disc. The patient's jaw deviated to the right because of pain in the temporal region. Other cranials were normal. The deep tendon reflexes were present and equal, the knee jerks hyperactive, the ankle jerks were rather sluggish. The abdominals and cremasterics were present, equal and active. Plantar responses were flexor. Astereognosis was not noted.

The white blood cells numbered 13,000; 74 per cent polys. Spinal fluid was clear, with a pressure of 180 mm. of water. Cell count showed 10 lymphocytes.

On September 16, 1937 the laceration had about stopped draining. The patient was operated on and an area of necrotic bone about the size of a ten cent piece was found beneath the laceration in the temporal bone. This was enlarged by means of a rongeur, revealing a small rent in the dura underlying it. The dural opening was enlarged and an abscess cavity opened into it. Foul-smelling thick yellow pus was evacuated and one Dakin tube inserted in the cavity. The wound was left wide open and packed loosely with iodoform gauze with wet Dakin dressing.

Postoperative Course. Following this procedure the temperature rose gradually to 101 on the third postoperative day. The general condition improved somewhat for three days. There was gradual diminution in the amount of drainage and the patient again became stuporous, at which time there was *no drainage* from the abscess.

I was asked to see the patient on September 21, 1937 and operation was done the same day, five days after the operation in which tubal drainage was employed. The area was dirty. There was a small crucial incision with flaps sutured together and a small Dakin tube protruding. The brain cortex had herniated to an elevation of about ¾ inch through the 1 inch cranial defect. Through this the tube protruded like a catheter from the cervix.

While the opening in the skull and dura were being enlarged to about the size of a fifty cent piece, a lumbar puncture needle was being inserted. The fluid was clear and colorless. After about 15 c.c. had been removed the brain hernia became softer, and the opening into the abscess larger. Thick yellow pus began to flow out. After the opening into the abscess was spread (firm adhesions had already formed between the cortex and meninges about the hernia) more pus was evacuated. This pus had been retained in three lateral pockets formed by collapse of the walls of the abscess about the rubber drainage tube so firmly that pus could not escape. A cross section

of the abscess would have been the shape of a three-leaf clover. After 45 c.c. of cerebrospinal fluid was removed, the abscess was wide open and measured about 2½ inches in depth and 2 inches in diameter. It had a smooth pinkish red lining about 4 mm. thick. An iodoform gauze "handkerchief" with inner iodoform strips was placed in position in the usual manner and a copious head dressing applied. Condition after operation was good.

On the second day postoperative the superficial dressing was changed. A spinal tap was done, 45 c.c. fluid removed and, after removal of the inner iodoform strips, a well-like cavity was seen. The handkerchief was not disturbed. The general condition was good. The herniation was controlled by frequent lumbar punctures. On the eighth postoperative day temperature was 100, and the pulse averaged 85. There was at this time only a dimple-like cavity about ¾ inch long, ⅝ inch wide and ½ inch deep. The wound had not been dakinized and there was some purulent reaction which is not seen in a well dakinized wound. Azochloramid was used because Dakin's solution was not available at that time.

On the tenth postoperative day all of the original gauze was removed, replaced in the usual manner, and dakinization was begun. On the fifteenth day, the temperature was 99, the pulse 82. The wound was granulating, and slight herniation was controlled by spinal taps, catharsis, enemata, elevation of the head and restriction of fluids. The patient was up and about the ward on the thirtieth day. He made an uneventful recovery.

The patient left the Jersey City Medical Center the day before Thanksgiving in 1937 and went to his home. He returned to his usual work in a gasoline station in February, 1938 and has worked there steadily ever since. He gets along without any trouble, has no complaints and does his regular work in the usual manner.

RESULTS

Of sixty cases of brain abscess of all types observed in the period from 1920 to 1939, seven cases were of traumatic origin. Three resulted from punctured wounds of the brain; one was produced by a thrown pair of scissors, one by a knife-blade producing a self-inflicted wound through a preëxisting cranial defect, and one by a broken-off knife blade which remained in the brain over twenty-eight years. Two resulted from fractures involving the mastoid in one instance and the frontal sinus in the other. One developed late after a self-inflicted gunshot wound of the skull and brain, a 45 caliber service pistol bullet inflicting the injury. One formed after the operative site of the compound fracture of the skull was well healed. All were males, ranging in age from 7 to 54. Four had convulsions before operation, and one, the patient with the gunshot wound, had

convulsions after operation. Seven were operated upon and all made uneventful recoveries.

W. GAYLE CRUTCHFIELD (Richmond): In the discussion of this subject, I believe that prevention should be stressed. There would be few brain abscesses of traumatic origin if patients with lacerations of the scalp and compound fractures of the skull received early treatment consisting of thorough debridement.

Dr. King's method of treatment and his meticulous postoperative care of patients speak for themselves. Such a high percentage of cures is an unusual achievement. We have used Dr. King's operation with a high degree of success but usually we have reserved it for those abscesses that fail to heal after continuous catheter drainage or repeated tapping.

Dr. King has not mentioned sulfanilamide as an adjunct to surgery. I am of the opinion that with the aid of this drug we may be able to obtain more cures with his as well as other methods of treatment.

JOHN RAAF (Portland, Oregon): I might paraphrase a statement by Osler and say, "From Macewen, who made one of the earliest contributions on the subject, down to the present time, the treatment of brain abscess has been one long traffic in hypophysis." Almost every surgeon who has treated a number of patients with brain abscess has devised his own method of treatment of the condition. Some surgeons advocate the multiple tapping method; some prefer to insert a small rubber catheter into the abscess cavity and allow the abscess to drain through the catheter; while others open the abscess cavity widely, view the interior of the cavity, insert hard rubber tubes, and pack about the tubes with gauze. Dr. King prefers still another method. I believe that those surgeons who are so attached to their own method of treatment of brain abscess and in effect say, "Come follow me and leave the rest of the world to its babblings," are assuming the wrong attitude, for I do not believe that every brain abscess can be treated by the same method. The selection of the method of treatment depends on the portion of the brain in which the abscess is located and the depth of the abscess from the surface as well as the length of time the abscess has been present.

Certainly there is something to be said for Dr. King's method of treatment. Nine days ago I was present at an autopsy upon the body of one of my patients whom I had operated on for brain abscess. The abscess had followed a frontal sinusitis and an osteomyelitis of the frontal bone. The abscess cavity was huge; I had opened it widely, sucked out the contents, inserted hard rubber tubes, and packed about the tubes with gauze. All had gone well for ten days, and then cerebrospinal fluid suddenly began to drain from the hard rubber tubes, and the patient subsequently died of meningitis.

One must remember that after an abscess cavity is evacuated, the wall of the abscess will advance upward; and if the tubes are not withdrawn

rapidly enough, they will push their way through into the ventricle as happened in this case. Dr. King's method of packing the abscess cavity with gauze would have been, I believe, preferable here.

I should also like to add my "amen" to Dr. Crutchfield's plea for adequate treatment of head wounds at the time of injury. Repeatedly one sees patients who have compound comminuted fractures of the skull which are inadequately treated. The attitude frequently taken is that the patient will die anyway so why bother with careful suturing of the scalp wound. As soon as the patient has recovered from the initial shock, he should be taken to surgery, and a thorough debridement of the wound performed. If this is always done, the wound will in a majority of instances heal by primary intention. This not only will eliminate the danger of subsequent formation of a brain abscess, but if at a later date some condition such as subdural hematoma is found to be present, the surgeon has a clean operative field in which to work.

JOSEPH E. J. KING (closing): I want to thank Dr. Crutchfield and Dr. Raaf for their very kind remarks.

I have not used sulfanilamide in cases of brain abscess since I have had no cases complicated by meningitis since the drug was brought out. Should I have to treat a case with an associated meningitis, I would surely use sulfanilamide. I have never resorted to simple tapping alone.

We have had what might be considered rather good results, but I do not insist on the sole use of the method which I have described. In my hands it has proved efficacious in most cases. We have had thirty-five patients who had, by any stretch of the imagination, some chance to recover. Some of the sixty patients were dead or moribund when I first saw them, and in a number, suppurative leptomeningitis had already developed, while in several more the lesions were multiple metastatic ones.

Thirty-one of thirty-five patients recovered, and four died. Three deaths are believed to have been preventable. One was due to a nurse's pouring eggnog down the trachea of a patient who was comatose, and she literally drowned. In another case the dressing was done by someone else in a distant hospital, and the bandage was removed by the patient who clawed into the brain substance and reinfected the brain. Another, a child, developed pneumonia due to having been placed by an open window in the winter, while dakinization was being carried out.

Dr. Crutchfield was very unassuming in his remarks. He and Dr. Coleman have had splendid results in a number of abscesses.

I could go into the different types of procedures, but time will not permit. The tube drain, of which Dr. Raaf spoke, has several disadvantages. It may become dislodged and slip out of the drainage tract, and when an attempt is made to replace it, it may be pushed into the brain substance itself and not into the tract. A secondary pocket may develop due to insufficient drainage of the abscess, and it may rupture into the ventricle in the same manner as an original abscess. Or, the tube may be held firmly in

place, and the distal end be thrust in bayonet fashion through the floor of the abscess into the ventricle. I feel sure that most surgeons dealing with brain abscess have had these experiences.

We have been fortunate in that we have not had a fatal meningitis with the exception of one case with gross rupture into the ventricle in an unmanageable patient. We have performed but a single operation for any given abscess, and have not had to resort to multiple operations or explorations.

In six cases in which colleagues had used a tube to drain the abscess, I have oserved that a small tube would be placed into the abscess cavity, a considerable amount of pus would drain out, and the patient would improve for about four or five days. Then the surgeon would notice that the patient was not "doing so well." When I was called to see the patient and removed the dressing, the end of the tube could be seen sticking up above the wound, in some cases with slight herniation of the brain, and not a bit of pus exuding from the tube. Nevertheless, the patient would be quite ill.

When the patient was placed on his side with the tube directed upward, and lumbar puncture was done, pus would begin to ooze out through the wound, both through the tube and alongside it, as the fluid was removed through the lumbar puncture needle. In most instances the amount of the cerebrospinal fluid removed would range between 35 and 45 c.c. By the time this amount was removed an enormous outflow of pus would take place. On exploration of the tract and dilatation of the external opening, one could observe that the abscess cavity was partially collapsed about the tube, leaving a pocket at thirds in the circumference of the abscess, so that a cross section of the abscess cavity and pockets with the tube in place would somewhat resemble a three-leaf clover. With removal of the cerebrospinal fluid, the cavity again became dilated so that only one cavity was present, and the pockets would disappear. When the cavity had opened widely, it was treated in the manner described, and in all six instances the abscesses healed.

THE USE OF ANTISEPTICS IN THE TREATMENT OF OPEN WOUNDS

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ANTISEPTICS may play a dual rôle in the treatment of an open wound: (1) their application to the skin about the wound to prevent further contamination of the wound by skin bacteria; and (2) their use as germicides in the wound itself.

Strong and convincing argument in recent years, notably by Reid and also by Koch and Mason has been advanced to indicate the harm that the indiscriminate use of irritating antiseptics in fresh wounds may cause in the prevention of rapid and complete healing. If soap and water and copious amounts of saline are used to irrigate carefully and thoroughly every portion of the wound, infection can be prevented and antiseptics in the wound itself are rendered unnecessary. Reid regards the fundamental concepts upon which proper treatment of the wound should be based as:

1. Hemorrhage is to be controlled.

2. Bacteria for the first six to eight hours are on the surface of the wound and are not invading and therefore simple mechanical irrigation will suffice to evacuate them.

3. Bacteria destroy nutritional support to and kill living cells.

4. Debris, clots, foreign bodies, and devitalized tissue promote bacterial growth.

5. Fresh wounds are rarely completely free or freed from bacteria. Their sterilization is relative.

6. Healthy living cells have great power to combat bacteria.

7. Healing occurs by growth of living undamaged cells.

8. Stimulus for growth of living cells is probably from the cell damage or chemical change in the wound.

9. Rest of the part injured promotes healing.

The important essentials in treatment are, therefore:

1. Control of hemorrhage.

2. Careful and thorough removal of debris, foreign bodies, clots, and devitalized and non-viable tissue (debridement).

3. Mechanical cleansing by irrigation, very thorough and gentle, to avoid damage to the remaining healthy living cells.

4. Few fine ligatures.

5. Splint to rest the involved part.

Treatment of an open wound should be inaugurated as soon as possible after the injury, certainly within six hours of the accident if infection is to be prevented. It should comprise:

1. Control of bleeding and protection of the wound. This can usually be accomplished in small wounds by covering the wound with sterile gauze and making pressure upon it. Large wounds may require for control of hemorrhage a tourniquet well above the traumatized area or a hemostat on the bleeding vessel.

2. Cleansing and sterilizing of the skin around the wound.
   This is best accomplished by:
   (a) Soap and water.
   (b) Alcohol.
   (c) Alcoholic antiseptic—tincture of merthiolate, or of bichloride of mercury.

Soap and water not only mechanically cleanse the skin, but have definite antiseptic power. When skin is relatively mechanically clean, alcohol and an alcoholic antiseptic may suffice. If soap and water are used, plenty of alcohol to dehydrate the skin should follow. If alcohol is not used, penetration of the antiseptic to the deeper glands of the skin is very slight and wound contamination from the skin is more likely. Experimental proof is not lacking to indicate that alcoholic solutions of antiseptics are more efficacious in destroying bacteria of the skin than watery solutions. Care should be taken that the skin antiseptic should not enter the wound.

3. Protection of the skin around the wound with sterile towels or covers.

4. Cleansing of the wound.

The wound should be very gently washed out with soap and water and irrigated with saline. All clots and foreign material—grit and gravel, and free debris—should be removed. Large bits may have to be lifted out with forceps. Cleansing should be done most painstakingly and methodically, beginning with the skin margins, then continuing with the subcutaneous fat and finally the muscle and fascia. All interstices of the wound should be reached. The opening in the skin should be enlarged whenever necessary to permit complete visualization of the wound. Finally, debridement of all devitalized tissue and dirt containing tissue must be thoroughly and carefully done, i.e., beginning with the skin margins, the subcutaneous fat, and finally proceeding to fascia and muscle. After debridement, the tourniquet, if used, is removed and all bleeding points ligated, and again the wound is thoroughly, gently, and

methodically cleansed with saline. (In laceration of the scalp with open fractures of the skull, diffuse saline irrigation is contraindicated; saline should be used freely, but only in small amounts because of the possibility of extensive flooding carrying infection to the meninges.) Only the finest ligature material should be used, whether silk or catgut, and as little tissue as possible should be included in the tie in order to avoid the pressure necrosis of ligature en masse that will cause breaking down of the wound. The manner in which all this is accomplished, how gently and thoroughly the procedure is executed, and not simply the procedure used, determines the success of treatment.

5. Drainage, closure, and splint.

In large wounds, drainage, particularly dependent drainage, is frequently indicated. Finally, the wound is sutured or packed wide open with vaseline gauze, or the Carrel-Dakin treatment is instituted, according to the specific indications in each case, and a snug but not tight bandage, splint, or cast applied to insure rest of the part.

COMMENT

Meticulous, careful and prolonged use of soap and water and saline will usually suffice to rid the wound of bacteria. These solutions are not destructive to the tissues, and make the use of antiseptics, as a rule, superfluous.

However, those who have had wide experience in the treatment of trauma have recognized the greater potentiality for and danger of anaerobic infection, notably with the gas or tetanus bacillus, presented by soil (particularly highly cultivated soil), contamination of wounds, in contrast to the usual industrial wound. Even in the face of the universal condemnation of antiseptics in the treatment of open wounds it seems to me that we should nevertheless weigh carefully the necessity and advisability of the use of antiseptics for this type of contamination. A case of tetanus (referred to our clinic), which developed four months after the original injury, antitoxin having been given, and the occasional case that has been reported in which tetanus has occurred in spite of adequate treatment of the wound and the prophylactic use of tetanus antitoxin suggest that in these soil contaminated wounds something more than the routine use of soap and saline, no matter how exhaustively and meticulously employed, is advisable. Most commonly these wounds are considered potentially liable to anaerobic infection and should not be sutured, but are best treated by the Carrel-Dakin method or by

packing wide open with vaseline gauze. It has seemed reasonable, therefore, if Dakin's solution is to be used in these wounds following the soap and saline routine that it would be logical to use Dakin's solution instead of, or following, saline in the actual irrigation of the wound as part of the primary treatment. Furthermore, Dakin's solution is a strong oxidizing agent as well as a powerful antiseptic and is easily obtained with proper pH and potency by simple dilution of the commercial concentrated solutions such as hychlorite or zonite. Certainly, if there is available a non-irritating and nontoxic antiseptic that would not damage tissue or alter in any way normal tissue repair it would be well to consider its use in these peculiarly contaminated wounds.

We have, therefore, in a small series of 150 cases, used Dakin's solution where gravel or soil contamination had öccurred, especially to ascertain (1) the incidence of infection after this treatment; (2) if delay in healing or breaking down of the wound resulted.

All these patients had injuries of sufficient magnitude to warrant admission to the hospital and remained under observation while healing took place. In only three did any infection occur, and these were all lacerations of the cheek into the mouth, a type of wound in which recontamination from the mouth is seldom avoided. In three some serum, and in two a drop of pus was noted about the sutures at the time of their removal. In five there was sloughing of a widely undermined skin flap, but without infection. There was no evidence in any that the use of Dakin's solution was responsible for delay in healing, but one wound with extensive tendon repair seemed to show more deep scarring of the tissues than might be expected if saline only had been used. Reid has noted in experimental animals, in comparing wounds treated with soap and saline with wounds in which strong antiseptics had been used, that in those healing apparently equally well, there was often a thicker and less pliant scar in the antiseptically treated wound. Carrell has shown that in an open wound in which one-half was treated with saline and the other half with Dakin's only a very slight retardation occurred on the Dakin side.

Twenty-five open fractures have likewise been treated with Dakin's solution for primary irrigation. Five of these were treated by packing wide open with vaseline gauze; ten by complete closure of the wound—all these were joint fractures; in nine the wounds were sutured and drained; and in one the Carrel-Dakin method was employed. In one of the cases where packing with vaseline was done there developed a slight superficial infection with

Staphylococcus albus, which did not influence the healing of the fracture.

From this very small series obviously no positive conclusion is justified, but these preliminary observations would seem to indicate that Dakin's solution does not interfere materially with healing and should serve as *an added safeguard* in the soil contaminated wound.

However, it is to be emphasized that the use of Dakin's solution or any antiseptic in the soil contaminated wound must be regarded merely as *an added safeguard.* The main essential in preventing anaerobic infection in these wounds is a thorough and meticulous debridement; i.e., removal of *all* the devitalized tissue, particularly devitalized muscle, the ideal pabulum for bacterial growth. Likewise, the prophylactic employment of tetanus and gas bacillus antitoxin is highly important—5,000 units of tetanus antitoxin instead of the usual 1500 will be more efficacious and this may be repeated in ten days in suspicious cases, as Garlock has suggested. As sulfanilamide has been shown to be effective against the gas bacillus, its routine use to prevent the possible onset of infection should have merit (Ogilvie).

DISCUSSION

There have been but a few investigations that bear upon the toxicity of antiseptics for tissue. Dakin found that bichloride of mercury, nitrate of silver, and iodine were all irritating to the tissues, but that Dakin's solution destroys *necrotic* tissue and does not damage tissue with a circulation. Smelo (1936) has reported merthiolate less toxic than phenol, iodine, mercurochrome and metaphen but not an ideal antiseptic because it is more toxic to connective tissue and epithelial cells than to bacteria. McClure has been interested to obtain an antiseptic, not destructive to tissue that could be used with tannic acid in the treatment of burns. He has reported the mercurials, bichloride, metaphen, merthiolate, as having a high toxic coefficient, with the cresols and hexylresorcinol relatively low. Meleney, for the anaerobically contaminated wound, has suggested the use of zinc peroxide where contamination with anaerobic and microaerophilic organisms was suspected or inevitable. The zinc peroxide, to be effective, must be applied as a creamy suspension in sterile distilled water to every part of the wound surface. When delayed closure seems advisable, the wound may be packed wide open with gauze saturated in zinc peroxide.

It would seem that this should be a fruitful field for further experimental investigation.

SUMMARY

Efforts to treat an open wound to promote rapid healing without infection must be directed first to the control of hemorrhage and then to the removal of all debris and devitalized tissue by irrigation with saline, but so gently, carefully, and thoroughly, as to wash out and rid the wound of every free necrotic bit, but not to damage the healthy tissue remaining. This is by far the most important element in the treatment. The most essential adjunct is debridement or excision of all the devitalized and contaminated tissue that is not free and cannot be washed out.

Antiseptics are of value in skin disinfection, but in the treatment of the wound itself are seldom necessary, and their indiscriminate use may harm normal processes of repair. An antiseptic, however, would be an added safeguard in anaerobically or soil contaminated wounds. In this category, it is suggested that Dakin's solution may be used. An ideal antiseptic for use in open wounds should possess effective bactericidal action and be nontoxic to tissues and not interfere with or delay healing.

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PRIMARY CLOSURE OF TRAUMATIC WOUNDS WITH ESPECIAL REFERENCE TO THE CONVERSION OF COMPOUND INTO SIMPLE FRACTURES

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I APPRECIATE the fact that in advocating the early closure of compound fracture wounds, I am opening up a controversial subject. The surgical profession is to some extent divided into two camps on the subject, one advocating the Orr or other forms of open treatment, the other side leaning to early closure of the wound with the idea of converting compound into simple fractures and avoiding in many cases, at least, prolonged drainage, chronic osteo-myelitis, sinus formation, disabling scars and other complications.

In 1924, Wilkinson[1] reported a series of fifty-four compound fractures in which primary closure had been done. Only 6 per cent became seriously infected. In 1929, I[2] reported the results in more than 100 cases of compound fractures treated by early skin closure in which satisfactory union was obtained in approximately 90 per cent. In this same communication I called attention to the value of liberating lateral incisions in dealing with certain of these cases in which excessive loss of skin or an unusual amount of swelling made it necessary to mobilize the adjacent skin to some extent in order to bring about closure of the wound without undue tension.

The neurosurgeons of this country have quite generally evolved a technique for dealing with compound fractures of the skull by the aid of local anesthesia, adequate antisepsis and asepsis with debride-ment, followed by closure of the wounds without drainage. Some of them are also recommending the use of relaxation incisions well outside the involved area so that a satisfactory closure can be brought about in cases in which there has been considerable loss of skin. Coleman,[3] Wilson,[4] and many others stress the danger of using drainage in such cases, especially if there is a concurrent injury of the brain, their belief being that the drain itself can and often does introduce infection, that it may create a sinus and be the cause of brain infection, edema and death.

The scalp, of course, has a rich blood supply and can thus over-come infection more readily than other parts of the body, yet it is

difficult for me to understand exactly why the difference of location should necessarily make such a vast difference in the preferred method of treatment. However, I will grant the premise that some
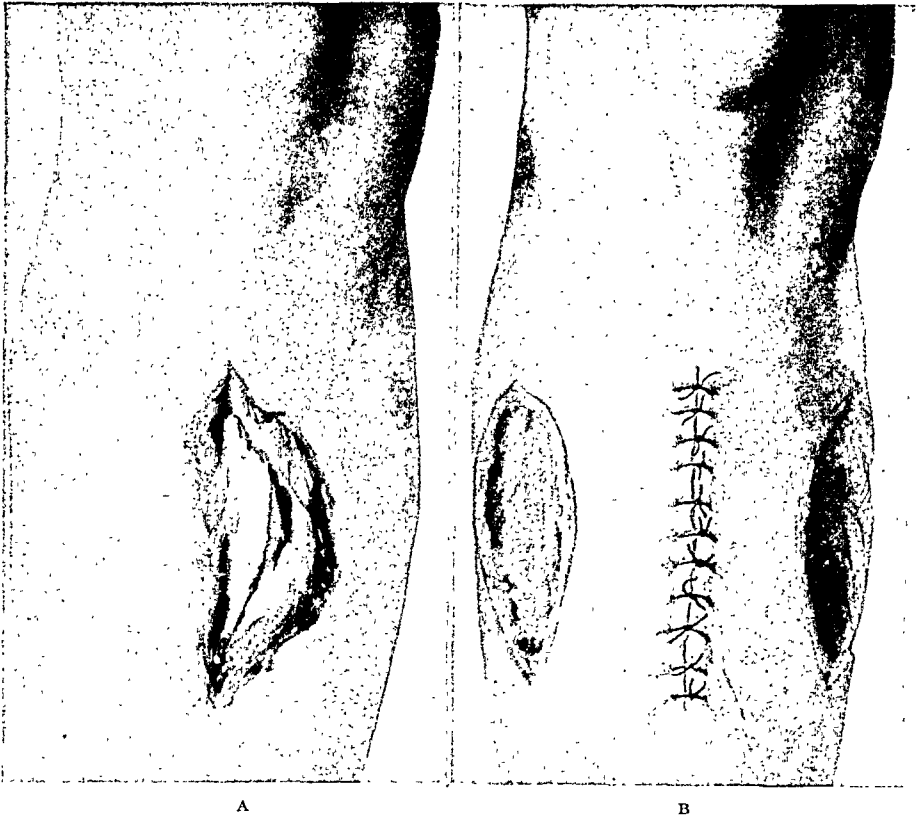
Fig. 1. A, compound fracture of tibia with extensive exposure of bone at point of fracture. B, showing liberating lateral skin incisions, allowing suture of skin wound over point of fracture. (From Cannaday, in *Ann. Surg.*, April, 1929.)

wounds may be so badly lacerated or so grossly contaminated that it would be foolish to attempt a primary closure; otherwise patients in a bad state of shock are not in condition for any prolonged operative procedure. It is also quite possible to secure good results by more than one method of treatment.

It is obvious that in conjunction with this method adequate immobilization is absolutely necessary. It will be argued that in some cases at least there will likely be an accumulation of serum in the wound, particularly when a certain amount of dead space is present. Most of the men quoted state that in such cases they practice loose closure in order that drainage by seepage or leakage may take place. This particular method of wound closure was practiced by Sir Arbuthnot Lane[5] in his no-hand-touch-technique for the internal

fixation of fractures of the long bones by the use of metal plates. The majority of the surgeons whose methods have been studied do not make a general practice of using drains.

It is taken for granted that the method described will be carried out in well equipped hospitals and by surgeons adequately skilled in the surgical technique and asepsis that should go along with any major operative procedure.

TECHNIQUE

The application of a first aid sterile compress of gauze which may or may not be saturated with an antiseptic solution is routine, while thorough emergency splinting of the part is, of course, necessary for satisfactory transportation of the patient. The methods in general use for the cleaning up of a compound fracture include the free use of liquid green soap and warm water. Shaving about the wound is followed by prolonged and thorough irrigation of the laceration with large amounts of normal saline solution. The skin about the laceration is painted with some antiseptic, the area is draped and an extremely thorough but conservative debridement is done, including grossly contaminated bone which can be pared away by the use of the chisel or rongeur. If possible the fracture is reduced, after which the skin wound is closed if it is practicable to do so, with due regard for the length of time which has elapsed since injury, the nature and amount of contamination, and the available skin present.

It is necessary that the debridement be carried out in meticulous detail, enlarging the skin opening if necessary for the proper inspection of all parts of the wound and for the purpose of extending the debridement into the ultimate recesses of the wound, and thoroughly cleansing and debriding contaminated bone ends as well. In case there is much dead space or likelihood of an accumulation of serum, the suturing is lightly done in interrupted fashion so that any accumulation of fluid can drain out by seepage between the sutures. In some of these cases, owing to excessive loss of skin or swelling, it may be necessary to make free liberating incisions laterally in order to be able to bring the skin edges together; likewise to allow adequate drainage from the wound by seepage underneath these skin flaps. Drains are dispensed with whenever it seems safe since they undoubtedly may introduce infection from the outside and predispose to sinus formation. During the process of debridement it is deemed advisable to redrape the area, likewise to change gloves and instruments, especially in dealing with the more extensive and

complicated wounds.* At this time the reduction of the fracture and the fixation of the limb must be brought about in a positive manner by plaster casts, splints, wires, pins, or other forms of traction and countertraction.

Quite a few surgeons practice keeping the wound covered with alcohol soaked or dry sterile gauze during the time in which the scrubbing and shaving of the skin surrounding the wound is taking place. The extremely thorough cleansing of the wound itself both before and after debridement with large quantities of saline is the method most favored at this time.

At the time of debridement, the skin edges and all badly damaged skin are removed, along with all lacerated or damaged tissue aside from major blood vessels and nerve trunks and tendons. Small detached bony fragments are removed, but larger fragments and especially those attached by periosteal strips are not removed. Soiled bone ends are debrided by use of a rongeur or chisel, with care not to sacrifice too much bone. Even simple punctures are explored carefully as they may contain foreign material. Bleeding points are ligated with fine catgut. After a painstaking debridement has been carried out, the wound is again irrigated with copious amounts of warm saline solution. Internal fixation of the fracture by metal plates, screws or wires is not generally felt to be advisable as it may lead to prolonged infection. Control of the fractured bones by skeletal traction is favored as being a comparatively safe and positive method.

Wounds which have been carefully debrided within six to eight hours after injury, may often be closed without drainage. Those treated eight and ten hours in many cases may likewise be sutured somewhat loosely to allow for possible wound drainage. Buried sutures are not used except for hemostasis and repair of tendons and nerves.

If a plaster cast is applied an adequate window should be cut over the wound for purposes of inspection.

A few illustrative cases are appended.

* Since March, 1939, we have been implanting sulfanilamide crystals in traumatic and other potentially infected wounds. We have observed a lower incidence of infection as a result of the use of this method. More recently we have also been using a 3 per cent solution of cocoanut oil soap in the cleansing of wounds, this being followed by free irrigation with normal saline solution. It is felt that this soap solution is distinctly bactericidal and not only helps to eliminate contamination of the wound received previous to operation but likewise to eliminate most of the wound contamination from the air-borne bacteria which are undoubtedly present in large numbers in all operating rooms.

CASE REPORTS

CASE I. A woman, 68 years of age, in crossing a dusty road in mid-summer was knocked down by a car. She received a compound fracture of the tibia and a simple fracture of the fibula. The tibia had broken through the skin and protruded for at least 2 inches. As her condition was good, she was at once given ether anesthesia, the wound was flooded with freshly prepared tincture of iodine followed by alcohol and then carefully debrided. The projecting end of the tibia which had apparently been grossly contaminated was pared away with a rongeur. The fracture was reduced and the wound was closed with silkworm gut sutures. These were allowed to remain in place for approximately two weeks. The leg was supported by a plaster cast with a window for dressings. Primary skin union took place. Bony union was delayed but took place in the course of four months.

CASE II. A mine foreman who had been badly injured in a slate fall was sent to me for treatment. He was not at all well at the time of injury and had had a persistent cough for some time past.

On examination he was found to have a simple fracture of the left forearm and left femur, a simple fracture of the fibula of the right leg and a compound fracture of the tibia of the same leg, with considerable loss of soft tissue over the anterior surface of the tibia at the point of fracture. He was in a state of shock and was treated for this. A dressing of gauze and cotton wet with merthiolate solution was applied over the wound. By the next day his condition had improved somewhat and at this time under local anesthesia, debridement of the compound fracture area was done. Rather long liberating incisions had to be made on either side of the leg in order that a satisfactory closure over the bone might be brought about. The fracture was reduced and the soft tissue wound closed with interrupted silkworm gut. The leg was held in position by a long posterior splint with a footpiece attached. After closure it was observed that each of the lateral incisions was approximately 4 inches long and that there was a bare gap about 1½ inches wide about the center of each lateral incision.

The operation proved to be successful. One month later the original skin wound over the fracture was perfectly healed. The granulating surfaces left by the liberating incisions had narrowed down to approximately ¼ inch in width at the widest point. For a time there was fibrous union of the fracture. In the course of three and one-half months firm union was present.

CASE III. In a compound fracture of the tibia and simple fracture of the fibula of eight hours' duration, the wound was cleansed, debrided and sutured under general anesthesia. However, it was closed with more tension than was desirable. Stay sutures of silkworm gut were used. A plaster cast with window was applied. Ten days later the wound broke down, but the walling off of the fracture had progressed so far that the bone was covered with healthy granulation tissue and no evidence of deep infection developed. Later the gap in the skin was covered by skin grafts. Firm bony

union took place without untoward event. It was obvious that the protection afforded by the skin, even temporarily, gave time for the building of a defensive wall at the site of fracture.

CASE IV.   A male, aged 25 years, was admitted to the hospital with a recent compound fracture of the right femur resulting from an injury when his right leg was caught between a mine car and a post. His doctor applied a long wooden splint to fix the fractured limb, dressed the wound and sent the patient to the hospital.

The patient was not in shock, the lower portion of the right femur projected more than an inch through the skin and was badly soiled with coal dust. X-ray revealed a comminuted fracture just distal to the trochanter with marked upward and outward dislocation of the shaft of the femur.

Immediate operation was done. In addition to the usual soft tissue debridement a careful paring of the contaminated end of the bone was carried out with the aid of a rongeur. A Steinmann pin was put through the lower end of the femur. The fracture was reduced and the wound was closed with interrupted sutures.

X-ray checks were made on the leg from time to time and while they showed a very slight upward displacement of the shaft, the femur outline was good and the position of the fragments such that a satisfactory result seemed likely.

The patient's temperature during convalescence went a little above 100°F. three or four times, but other than that it remained normal. When he was discharged from the hospital he walked with the aid of crutches which were later replaced with a cane. He was able to return to work five months after discharge.

CASE V.   A child, 6 years of age, was admitted to the hospital with a compound depressed fracture of the skull over the frontal parietal area. About one-half hour after the accident, the wound was debrided and sutured. It was found that the dura had suffered a moderate laceration, but the surgeon did not consider it necessary to apply a fascial graft. A small amount of traumatized brain tissue was removed. The wound was debrided and closed with black silk sutures. It was inspected from time to time and remained apparently clean. Seven days later, when the patient's head was being dressed, it was noticed that there was a serous discharge from the wound, that the skin edges had separated and that brain tissue was definitely bulging through the wound to a level of at least $\frac{3}{4}$ inch above the pericranium, also that the pulsations of the blood vessels were pushing this hernia up and down in a most alarming manner.

I saw this case in consultation and advised that another debridement and a secondary closure be made. The child was again anesthetized, the brain hernia was scooped out with the electrosurgical unit and the skin edges freshened. Since the retracted skin edges would not meet, free liberating lateral incisions were made, after which the wound was closed with sutures of black silk and the lateral incisions were packed with iodoform

gause. Healing took place without untoward incident and the result has thus far been satisfactory. No apparent bulging has occurred.*

The subject of compound fractures is of such great importance it is worthwhile to present the collective views and methods of a number of surgeons who treat considerable numbers of such cases. Letters of inquiry have been sent out to a number of these men, to determine whether or not they favor debridement and primary closure of wounds, especially in those associated with compound fractures. We find that the majority of them do favor this method of treatment. A very few, Sherman[6] and Campbell,[7] apparently favor leaving practically all compound fracture wounds open. The replies have been abstracted below.

Sherman favors dakinization followed by secondary closure when practicable.

Graham[8] says that he has definite figures on eighty cases treated by primary closure, at the Memorial Hospital in Richmond, thirty in one series and fifty in another. He believes that there have not been more than four serious infections in the entire group.

Cotton[9] states that loose closure with interrupted sutures which allow drainage by leakage rather than the installation of a drain frequently offers the best solution of the problem, but that a rigid technique is very necessary to the success of the method.

Pfeiffer and Smyth[10] say that immediate closure should not be attempted unless the surgeon is capable of carrying out an extremely careful technique. Naturally the degree of contamination and the amount of damage that has been done to the soft tissues would have a great bearing on the outcome.

Baldwin[11] favors primary closure, practices posterior or gravity drainage and uses tincture of iodine to disinfect the wound. Sometimes he irrigates with a dilute iodine solution followed by saline.

Swart[12] has been making use of primary closure in compound fractures for sometime past and finds that the results from the standpoint of primary healing are reasonably satisfactory and that in a good many instances, a slight wound infection may not seriously interfere with early and satisfactory union. He is of the opinion that the method is well worthwhile.

Anderson[13] uses closure in most of the cases that come from the mines if he sees them early. Cases that are badly contaminated he leaves open. He uses normal saline to cleanse the wounds.

* This case is from the neurosurgical service of Dr. Archer A. Wilson who made the final closure.

Scott[14] has been continuing the technique of careful debridement and primary closure for several years and has found it very satisfactory. His percentage of infection is low (he would estimate it to be less than 10 per cent). He does an extensive debridement and washes out the wound with large amounts of normal saline.

Estes[15] believes, as no doubt we all do, in treating each case on its own particular merits. He feels if the outcome of the case is doubtful it is better to leave the wound open. His decision to close or not depends greatly on the extent of muscle damage that is found, irrespective of the wound through the skin. When there is extensive muscle damage he does not close the wound primarily. A wound that has been contaminated with soil he does not close unless he feels that the debridement has been very satisfactory; usually he treats the wound in such cases by the Carrel-Dakin method. In general, he says that he seldom closes a compound fracture of the humerus or femur but is more apt to close a fracture of the tibia in which there has been little muscle damage, or fractures of the forearm in which there has been little or no injury to the muscle. He favors extensive irrigation and exploration of the wound, incising the skin if necessary, and following this in many cases by the use of Dakin's solution. Thorough debridement of the entire area is followed by a search of all the interstices of the wound and further irrigation with Carrel-Dakin solution, especially for the large wounds. Small wounds he may pack with vaseline gauze. Superficially situated wounds associated with compound fractures he may close with loosely tied interrupted sutures.

Rankin[16] tries to carry out a thorough debridement and follows this by closure, especially in cases that have been received within six hours of the accident. He expresses his opinion that 90 to 95 per cent of these cases heal by primary union. He, of course, excepts cases that have had soil contamination. After six hours he thinks considerable judgment has to be used and if the injury has gone for more than twelve hours, he thinks it should usually be treated by the open method. He has no hesitancy about using internal fixation, if necessary, for early fractures. In cases that are seen two or three days after the accident occurs, he considers it very dangerous to attempt manipulation.

Funsten[17] has treated more than 300 cases of compound fracture in the past six years and has closed most of them after extremely thorough debridement. He fells that the percentage of seriously complicating infections will not reach more than 8 per cent, perhaps less. In cases that are seen late and in some where the usual complete

debridement is handicapped he continues to use drains, but removes them in forty-eight hours through windows in the casts if the temperature is normal. His percentage of gas bacillus infection has been very small, perhaps partly due to the efficiency of the gas bacillus antitoxin.

Stallard[18] continues to close early compound fractures after debridement, using interrupted sutures loosely placed with a window cut in the cast for dressings if necessary. He also places his patients on prophylactic prontylin therapy to counteract the possibility of infection.

Byars[19] practices early closure of the soft tissues associated with fractures of the face and uses drainage if the condition of the wound seems to indicate it. Wounds of the floor of the mouth are never tightly closed. He says that some of them are packed with gauze; in other cases an opening for drainage is made underneath the chin. Lacerations under the mandible associated with fractures are closed early but drains are inserted to the fracture line. In other words, in a wound so situated as to be contaminated by mouth secretion, he uses dependent drainage if possible.

Bailey[20] considers every compound fracture case an individual proposition and does not desire to close such a wound if it has been exposed to an environment that might encourage severe infection. He often closes the wound that permits careful scrutiny and where antisepsis may be applied. He fears the tendency to pull too far toward the radical closure of preinfected wounds.

Outland[21] uses the Böhler technique in the treatment of compound fractures. This essentially consisting of immediate complete debridement followed by closure of the skin alone, no deep sutures being used except in case of nerve or tendon lacerations. A tube drain is inserted to the depth of the cavity about the fracture and passed through a small stab wound away from the original wound. He believes that this insures drainage in case of infection and also relieves tension on the suture line. The drain is removed in forty-eight hours if no sign of infection is present. He states that his real difficulty is with the small puncture wound associated with fracture. The question of whether to debride or not to debride he usually settles by merely applying a sterile dressing. In such cases he feels that if surgical operation is undertaken, the incision should be enlarged and a complete excision of the damaged tissue done.

Garr[22] as a rule closes primarily compound fractures in which there is no excessive damage to the skin and soft parts. He thoroughly debrides the wound, carefully cleansing about it with ether,

alcohol and tincture of merthiolate, puts in interrupted sutures rather far apart to allow free outlet for any seepage that may develop. He does the same thing in larger wounds but usually puts in a small wick of vaseline gauze down to the site of fracture which is removed in a few days if infection does not develop.

Prillaman[23] debrides and closes most compound fractures, installs Carrel-Dakin tubes (usually one or two) beneath the suture line and irrigates the larger wounds with Dakin's solution. When the temperature indications are favorable the tubes are removed.

Vance[24] believes that soft tissue wounds associated with compound fractures that are not too severely traumatized can be debrided and closed. If there is a very great loss of soft tissue and it does not seem possible to do this well he leaves the wound open. If wounds are very small he usually does nothing to them at all.

Carothers[25] makes it a practice to close the wound wherever possible, providing, of course, the case is seen within a reasonable length of time. He considers eight hours about the longest time after which immediate closure can be carried out. He makes relaxing incisions in the skin on one or both sides, if necessary, in order to get a complete closure of the skin over the point of fracture. If relaxation incisions are made immediate Thiersch grafts are applied to the newly exposed raw surfaces. In case of badly lacerated wounds after debridement he closes loosely so as to allow drainage. His luck with primary closure of wounds in compound fractures has been so good that he believes in carrying out this procedure whenever it is anatomically possible, if the case is seen within eight hours. He cleanses the wound thoroughly with saline and even if there is no fracture he splints the limb to cut down the trauma of movement. In compound fractures, in addition to splinting, he usually uses two pin traction.

Strickler,[26] in handling compound fracture cases seen early, usually does a thorough cleansing followed by a careful debridement, sutures the tissues loosely and occasionally uses a small rubber tissue drain. He does occasionally pack the wound with vaseline gauze after cleansing and debridement. His results in both the procedures have been about the same, but of course wounds packed with vaseline gauze require a very much longer time for healing.

Brenizer[27] favors primary debridement followed by secondary closure about a week later if no infection is present.

Boland[28] says in regard to the primary early closure of soft tissue wounds associated with compound fractures his rule has been in small wounds to cleanse, but make no attempt at closure. In

large wounds a thorough cleansing is done and the wound closed after thorough debridement.

Sullivan[29] believes in individualization and careful discrimination. His tendency is toward primary closure after chemotherapy and careful removal of traumatized tissue. If excessive tension is found, especially in the skin suture line, he attempts to relieve that tension by lateral incisions with elevation of the flaps for the purpose of covering the fracture as well as providing drainage for the injured area.

Where contamination by foreign bodies such as grease, clothing, dust, etc., is evident, closure of the wound is delayed until no evidence of infection is present, then secondary closure is made. Proper attention to the fracture by skeletal traction and fixation by plaster or splints is carried out in detail. The nature of the treatment of compound fractures demands proper hospital facilities for a successful outcome. Under less favorable environment he believes in reducing the fracture and maintaining reduction as best can be done at the time and leaving the wound open.

Trout[30] believes in individualization, thorough cleansing, thorough debridement. His general policy is to close incisions without drainage.

Stack and Magnuson[31] cleanse the surrounding skin with soap and water after covering the wound with sterile gauze dressing. The skin around the wound is shaved and cleansed again, after which the wound itself is thoroughly cleansed. A careful debridement including the skin edges is done and this is followed by irrigation of the wound with normal saline. The wound is then closed and the fracture treated as a closed fracture. If during the course of the operation they feel that any instrumental manipulation of the fragments will enable them to bring about a proper realignment, this is done with minimal trauma. They do not use internal fixation during this phase of treatment. They believe that the closed method of treatment is usually successful if carried out within a period of eight hours. After this safe period the same procedure is done, but the wound is closed quite loosely and warm moist dressings are applied, secondary closure being contemplated when the absence of infection is assured. Older wounds in which frank infection is already present, are treated in the usual open manner, either by irrigation or by the Orr method, depending upon which is deemed more suitable.

Battle[32] favors primary closure after debridement followed by the immediate application of a plaster cast.

Caldwell[33] favors early and thorough cleansing and debridement, including the removal of soiled bone ends. He advises against pouring strong antiseptics into the wound. If the case is seen early and the wound can be closed even, by making parallel releasing incisions, this is done. He feels that in case of extensive crushing injuries of the thigh muscles associated with fractures, the Carrel-Dakin treatment is preferable. He favors the Orr method in the more serious and highly contaminated cases.

Henderson,[34] in speaking of the treatment of wounds, makes the following statement: "One of the most important factors in the treatment of this type of wound is the cleaning with green soap and water. Scrubbing with green soap and water is of more benefit than are the chemical solutions." As to which wounds are to be closed without drainage, this, in his opinion, depends somewhat on the amount of time that has elapsed between the accident and the arrival of the patient at the hospital. After a number of hours have elapsed between the accident and the dressing most wounds are infected and drainage should be instituted. For most wounds, particularly those around the joints, the part should be immobilized.

Goodman and Reeves[35] advocate closure of compound fractures, especially in cases seen from six to eight hours following injury. In this classification of cases they do a complete debridement and do not drain as a rule.

Reid[36] has stated his conclusions following a special study of the subject of the treatment of open wounds. He says that for all practical purposes, up to about eight and one-half hours after the receipt of the wound, infection can be considered to be on the surface. After that the penetration will gradually go in deeper. He considers a thorough debridement under most scrupulous aseptic precautions to be absolutely necessary in such cases. Again he stresses the importance of closure without undue tension, the avoidance of strangulation of large blocks of tissue by ligation and the use of fine suture material rather than coarse in ligating blood vessels.

Golden[37] advocates a modified Orr treatment for most compound fractures, and thinks that other factors other than the absolute length of time have to be considered.

Ralsten[38] cleanses the skin thoroughly with green soap and water, using a scrubbing brush when necessary, and rinsing with ether. The wound is cleansed thoroughly with gauze and saline solution. The wound and adjacent skin are then painted with an antiseptic, usually merthiolate or mercresin and the field draped. A debridement is

done, with an attempt to stay on the conservative side and at the same time remove all soiled and devitalized tissue. Accurate hemostasis is secured at this time. The wound is closed without drainage and fairly snug dressings are applied. If a fragment is protruding, it is subjected to the same treatment as the soft structures. This author does not hesitate to apply an antiseptic to the wound or even to pool it there for four or five minutes before beginning closure of the wound. In some of his compound fractures he resorts to internal fixation, using vitallium plates. He states that in about 80 per cent of his compound fractures in which internal fixation has been done, he has had no infection and has secured good union. Although in some of these, signs of foreign body reaction developed after four to ten weeks and the plates and screws were removed, without such definite indication the plates were not removed. He believes in applying the above technique, especially in cases received in the hospital during the first eight to ten hours. In wounds received after this interval or in those patients whose condition upon admission is too critical for any kind of surgery, he allows the wound to remain packed open for a few days and then performs secondary closure if there are no signs of infection.

In some compound fractures in which the wounds were very extensive he performed the cleansing and debridement as mentioned, followed by reduction of the fracture. He then packed the wound open with vaseline gauze and applied a plaster cast in accordance with the Orr method. All these cases have eventually developed some infection and subsequent chronic osteomyelitis, but nothing of a severe type.

McMaster,[39] in discussing his technique, says: "The skin is washed with soap and water, grease is removed with benzine, and the hair is shaved, during which time the wound is covered with an alcohol-soaked or only sterile gauze." One of the standard antiseptics may then be applied to the skin, or as advised by Koch, the skin is again washed and cleansed with soap and water. He advocates primary closure in the case seen early, with the Orr treatment reserved for the cases that have gone for a longer period.

Kennedy[40] believes that sulfanilamide is of great value in the treatment of infected compound fractures. According to him, it was first used in this class of cases at Johns Hopkins Hospital in January, 1937, on a patient with a definite gas bacillus infection of a compound fracture. In the treatment of compound fractures fixed traction must be applied as soon as possible. Thought must be given to life and limb—and life comes first. In the limb we think of the fracture and

damage to the soft parts, and the soft parts come first. It is important that the patient be in the hospital not later than six hours after the accident, if possible, to avoid infection. During the cleaning up and debridement of the wound, he believes in changing drapes and instruments. He uses tetanus antitoxin routinely and also polyvalent antitoxin for gas gangrene.

Mock[41] believes that it is impossible to make any hard and fast rule regarding early closure associated with fractures. In some of his cases early closure is done while in others treatment is managed as for infected wounds, with delayed closure. However, most of these last granulate and heal without being sutured. He says that, from a practical standpoint, early closure of the wounds cannot well be carried out if the patient is suffering from shock. In such cases it would obviously be dangerous to attempt immediate debridement and closure.

Mock also feels that if the patient gets immediate first aid with proper splinting of the fracture and arrives at the hospital in good condition, there is a much better prospect for immediate closure from the standpoint of both shock and infection. On the contrary, in many instances where the patient is mishandled from the start, there is considerable delay before he receives any intelligent medical care and a large area of contamination is present, the situation is entirely different. He feels that from a practical standpoint it would probably be safer for the average man to leave the wound open. However, he has some doubt about the advisability of using a vaseline gauze pack and thinks it may add to the chances of infection in the wound. He favors secondary closure in some cases. In bad injuries the question of saving life must come first, and therefore debridement and closure may have to be delayed.

Emmett and Hawkins[42] have been practicing early debridement and closure for a number of years and find the method satisfactory in most instances.

Whitman[43] advocates and practices a modified Orr plan in dealing with compound fractures. If the wound has been made from within out, he debrides and sutures, but does insert a vaseline gauze drain and applies a cast which he does not remove or disturb unless there is an elevation of temperature persisting for more than five days. Ordinarily the gauze drain is removed through a window in the cast about the fourth week. In compound fractures in which the laceration of the soft tissues has been made from without, the wound is left more widely open and more extensively packed with vaseline gauze. If skeletal traction is used instead of a cast, a dressing is usually

applied over the wound. If mutilation of the soft tissue is extensive, Dakin's irrigation is used.

<div align="center">COMMENT</div>

Some of the advantages of the primary closure method are as follows: First, the patient is saved in many instances a prolonged period of hospitalization, as well as the pain and inconvenience of frequent dressings. Second, the injured limb, after healing, is left in a much better condition; if the case turns out successfully, it is covered with relatively normal skin rather than a large area of scar tissue of low vitality that is prone to break down on slight provocation. Third, the loss of time and the expense of care can be kept down to a reasonable minimum.

Summing these advantages up: (1) the patient is often saved prolonged hospitalization and discomfort and expense; (2) the doctor is saved time utilized in applying numerous dressings; (3) the hospital is saved many patient days, much of the expense of prolonged hospitalization and the cost of many dressings; and last and most important (4) the injured part of the body is usually left in a much better condition, being covered with normal skin rather than with scar tissue of low vitality.

However, the technique can only be carried out successfully by a surgeon who has thorough training in this class of work. It requires a most scrupulous asepsis and meticulous attention to detail.

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DISCUSSION OF THE PAPERS OF DRS. ESTES AND CANNADAY

MONT R. REID (Cincinnati): Before I make any comments on these two splendid papers, I want to express my appreciation for the privilege of attending and participating in this first meeting of the American Association for Traumatic Surgery, which I feel is going to be a very distinct contribution to surgery in this country. If it were possible. I am sure that the spirits of Paré and Billroth, of Sir Astley Cooper and Lord Lister would really rejoice at the formation of a society for the study of wounds. In a sense, I think a synonymous term for this Society might be the American Association for the Study and Treatment of Wounds.

After the work of Lister and the twenty-five years following that, when the fundamental principles of asepsis and antisepsis were being established, many people had the feeling that the problem of treating wounds had been solved. The men whom I mentioned felt that the proper treatment of wounds was the most important requirement of the surgeon. I feel that that is equally true today and that it is always going to be true so far as surgery is concerned.

Many surgeons have come to deplore the lack of interest during the past generation in this very fundamental problem, and I think that that feeling is responsible for the revival of interest in wound healing. An

unfortunate thing during this period of lack of interest in the treatment of wounds was that most of us became more or less the victims of routine and of the propaganda of the manufacturers of germicides which stressed the points that asepsis and antisepsis were the only important factors in wound healing. So I am extremely happy to be here on this occasion, because I feel that your society is going to be a great influence in revaluing all of the important factors involved in the problems of wound healing.

I said in the beginning that before I commented on these two excellent papers I wanted to make a few general remarks. Praise is now my only discussion, because I find no points of disagreement. I have enjoyed the papers tremendously, and I think they have stressed the fundamental principles of wound healing.

In Dr. Estes' paper he spoke of the methods of controlling hemorrhage in traumatic wounds, such as pressure with gauze. He does not resort to the use of a tourniquet unless it is necessary. To his advice I would like to add the simple expedient of elevating the wounded part. Often in the teaching of students, or even of interns who become excited in the management of a wound and put on a tourniquet to control the bleeding (often ineffectively if there are two bones at the site of the tourniquet), I make them look foolish by removing the tourniquet and holding the hand or foot straight up in the air and having the bleeding stop almost immediately. A great many doctors do not realize the value of an elevated position in the control of hemorrhage from wounds.

I think Dr. Estes has drawn attention to a very important point by warning us not to let our great zeal for cleaning and closing traumatic wounds be extended to certain wounds which should never be treated in that way. Certain wounds can certainly be so badly soiled that it is unwise surgical judgment to endeavor to close them even after the most meticulous cleansing and debridement.

That idea brings me to the discussion of another point. So many people have the idea that if a wound is not to be closed there may be a relaxation in the meticulousness of the toilet of such a wound. I feel that such ideas are very erroneous and that such practices can in no way be justified. A complicated wound which is not going to be closed deserves just as meticulous debridement, washing and cleansing, as does a wound that you are going to close. Theoretically, it deserves more, for it will not have the protection of a skin covering.

After that is done, it has been my general policy and feeling that such a wound should not be disturbed until the protection of granulation tissue forms in it. After all, lacking skin or mucous membrane, granulation tissue is the next best protection of any wound, and, personally, I think it usually forms most effectively when the wound is least disturbed.

I do not feel competent to discuss compound fractures. I think Dr. Cannaday has brought out some very excellent points and has also stressed the important fact that we should not let our enthusiasm run away with

judgment in our revival of interest in the cleansing and closing of compound fractures. Certain cases will be so badly soiled that no attempts at closure should be made even after the most careful debridement and cleansing.

He brought up one procedure which we have not used very much and that is the use of rongeurs to cut away dirt-stained periosteum and bone. We have felt that it was safer to use a very sharp chisel and a hammer rather than the rongeur, which often leaves fragments of bone and may grind dirt and organisms into the freshly cut surface of the bone.

I repeat again that I have enjoyed the privilege of listening to your papers and of participating in your meeting.

PHILIP H. KREUSCHER (Chicago): I was very happy to hear this paper and was particularly interested that facts were brought out which evidently had been lost in the last few years. So many of us have forgotten our experiences during the World War and the methods of treatment which were finally practiced during the latter part of the reconstruction period.

We all remember the thousands of men whose wounds became infected and have not forgotten the morbidity and mortality which followed these infections. The institution of the Carrel-Dakin method of treatment and their management of wounds revolutionized the treatment of sepsis and saved many thousands of extremities and many hundreds of lives. The results were so good that the surgical staff of the entire army of France, England and America expressed themselves satisfied with the fact that the discovery or the introduction of the Carrel-Dakin method of treatment was the greatest advance of wound healing since Lister and Pasteur.

We all began using the Carrel-Dakin method of treating wounds and became very enthusiastic for a time, but most of us discontinued its use because there was so much difficulty in preparing the solution, there seemed to be so much irritation of the wound and the interns and nurses, because of the more or less complicated method of management, grew tired in their efforts to carry it on. There are a few men who have never entirely discontinued this method. Among them is Dr. William O'Neill Sherman of Pittsburgh, who was the first to introduce it in this country and who spent many months with Carrel in France.

At the present time a great many of the objections to this method of treatment have been removed. Hypochloride, which is 4.05 per cent sodium hydrochloride, is now on the market and has been for years. One can simply take this solution and dilute it with seven parts of sterile hypotonic salt solution or with sterile distilled water, and it is ready for use. This solution is quite stable and can be kept for weeks in a dark container in a cool place without losing its efficacy.

For two and a half years I have used this method of treatment in a large number of cases. At the dressing stations in the steel corporation emergency hospitals we give approximately 12,000 treatments a month,

90 per cent of which are for acute wounds from abrasions, cuts, etc., and acute compound fractures.

The subject of careful preparation of the tissues around an open wound is very important. A great difficulty is to persuade the nurses and assistants that it is necessary to clean the surface of the skin around every wound and abrasion, that this must be done very thoroughly, and that it must include not only the shaving of the hairy surface but also a very thorough cleansing of otherwise normal skin.

Primary debridement of the wound itself, especially in cases with compound fractures or deep lacerating wounds, is very important. It is so well understood that it is not necessary for me to go into it any further. Several sets of instruments should be used in cases of this type, especially in the very dirty wounds.

I am glad to see that we are coming back to the rational treatment of wounds and that we are beginning again to make use of the things which science has placed into our hands rather than looking about for false gods.

W. L. ESTES, JR. (closing): I have nothing further to add except to thank both Dr. Reid and Dr. Kreuscher for their delightful discussion as they have brought out only too well the points that may have been glossed over in the paper.

JOHN E. CANNADAY (closing): I have nothing more to add except to express my appreciation for the illuminating discussion and to thank those who took part in it. I did mention the use of the chisel in debriding bone. I think it definitely has its place.

SURGICAL TREATMENT OF INJURIES OF THE CHEST

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INJURIES to the thorax, to the viscera within and contiguous to its walls, are surgical problems for accurate diagnosis and adequate treatment.

Adequate treatment in a well equipped hospital with a completely organized competent, experienced surgical and nursing staff is vastly different from what is proper treatment for the same type of case in the country or in a small hospital with meager facilities and short handed personnel.

These are important factors which determine whether radical or conservative measures should be immediately invoked. Limitations imposed by a lack of personnel and facilities to cope with exigencies that might arise during treatment should govern the plan of procedure. The objective of any plan of treatment is to restore the altered conditions to as near their normal status as possible with the least added surgical trauma.

In the early period of the World War injuries to the thorax were attended by a high immediate mortality. Complications with later death or chronic invalidism was the rule in the cases which did not promptly succumb. During the later period with better organization of surgical personnel and hospital facilities two plans of treatment were devised, one by the British[1] and French[2] was radical; the other by the Italians[3] was conservative. From these plans, principles of and indications for surgical treatment of chest injuries were crystallized which greatly reduced mortality and complications.

Circumstances attending and the injuries sustained by the chest, in civil life differ greatly from those inflicted in war. The principles of treatment do not differ. Casualties in civil practice are usually multiple, those of the thorax commonly of the closed type. The extent of internal damage in closed injuries of the chest is more ·difficult to estimate than that in open chest injuries. They demand a high degree of surgical judgment in selecting the opportune time and proper procedure for their treatment.

Injuries of the thorax may be classified as:

Closed {
Contusion of the extrathoracic structures.
Contusion, compression of the thoracic wall; with or without direct or indirect fracture, dislocation of ribs, costal cartilages and sternum.
Contusion, laceration of the parietal or visceral pleura.
Contusion, compression of intrathoracic structures.
Laceration of the heart or great vessels, intra- or extra-pericardially.
Contusion, compression of the lung, trachea, bronchi.
Contusion, laceration of the diaphragm, subdiaphragmatic, viscera the liver, spleen, kidney or stomach.

Open {

External {
Laceration of extrathoracic structures to the pleura: Compound fracture of ribs, cartilage, sternum, with and without penetration of the parietal and visceral pleura.
Large or small direct open wound in the thoracic wall.
Laceration or perforation of the external wall, pleura, lung, bronchi, trachea, pericardium, heart, diaphragm and subdiaphragmatic viscera.

Internal {
Contusion, rupture, laceration of lung, bronchi, trachea, mediastinum, diaphragm without an open defect in the thoracic wall.

The degree of damage depends on the character, direction, intensity and duration of the traumatizing force, the elasticity of the chest wall, ribs, cartilages and sternum, the nature and extent of the injury to the parietes, the type and location of trauma to the intrathoracic viscera, the mediastinum and its contents, the diaphragm and subdiaphragmatic viscera.

Injuries are more serious in the upper than in the lower thorax, more urgent the nearer the root of the lung. Injuries to the heart, the great vessels, trachea and mediastinum are most serious and frequently promptly fatal. In open chest wounds the gravity increases with the size of the opening and the length of time it remains patent.

Blunt traumatizing compressing force, especially on elastic chests of the young, can and often do cause extensive or fatal internal injuries without the slightest evidence of trauma to the superficial structures. The visible trauma to the thoracic wall is no index to the character or degree of intrathoracic damage.

Immediate collapse or sudden death may ensue from compression of the chest. This may be due to shock, vagosympathetic reflex, vascular paralysis, traumatic angina pectoris with acute cardiac insufficiency and consequent cerebral anemia.[4,5]

In cases surviving the immediate effects of trauma, there are the dangers of acute massive, continuing, recurrent or secondary intrathoracic hemorrhage, delayed rupture of the myocardium or great vessels, open, closed or tension pneumothorax, massive collapse of the lung, paradoxical interpulmonary breathing, emphysema of the mediastinum, extra- and intrapericardial tamponade, and the late hazards of infection; bronchopneumonia, traumatic pneumonitis, abscess, gangrene of the lung, empyema and cellulitis of the mediastinum.

The first examination of patients with chest injuries should be brief, gentle and as thorough as possible. Avoid all unnecessary moving and shifting of the patient. An increasing hyperresonance indicative of tension pneumothorax or a mounting dullness suggestive of intrapleural fluid should be promptly investigated by a diagnostic aspiration of the pleural cavity through a small gauge needle. This will give more reliable information with less discomfort and danger to the patient than prolonged, painful examination. During the first twenty-four to forty-eight hours the progress of the intrathoracic changes can be more accurately observed if the chest is not strapped with adhesive. An almost equal degree of immobilization and comfort can be secured by applying a firm wide chest binder without rolling or disturbing the patient. The binder can be conveniently opened and closed as often as reëxamination demands.

X-ray examination is indispensable. In the initial stage *x*-ray is not always conclusive as to the nature or extent of the damage. In the severely injured the moving and shifting necessary to secure proper exposures for reliable information increases shock and aggravates the damage already sustained. Fluoroscopy is more conclusive than *x*-ray films in the diagnosis of intrapericardial tamponade, the absence of expansile cardiac pulsation being pathognomonic. The most informative films are those taken in the upright or erect positions. The erect posture is inadvisable during the first twenty-four hours. Carefully elicited physical signs give sufficient information for early treatment in most patients who survive transportation and admission to the hospital.

Reaction to injury varies greatly with different individuals. The reaction is much more severe in those who have had previous pulmonary or cardiac lesions.

Pain is greater in parietal trauma than it is in much more serious visceral damage. The intensity of pain is no reliable index to the gravity of internal injuries.

Respiration in chest injuries is guarded. Inspiration is shallow and quiet, expiration forced and audible. Contractions of the diaphragm, painful splinting of the abdominal muscles and acute ileus especially in trauma of the lower thorax may lead to the erroneous diagnosis of subdiaphragmatic damage.

Dyspnea may be severe and immediate due to pain, irritation of the pleura, or aspiration of foreign bodies from the mouth, i.e., gum, tobacco, candy. Dyspnea from massive collapse of the lung in wide open pneumothorax is prompt, increasing in severity with the size of the open defect in the chest wall or large bronchus. Tension pneumothorax causes a progressive dyspnea. This progression is directly proportionate to the increase in intrapleural pressure and consequent mediastinal flapping and shift to opposite side. Other causes of dyspnea are anemia secondary to massive hemorrhage, waning cardiac capacity, cardiac tamponade, splanchnic vascular dilatation, acute ileus with distention of stomach and bowels. Late dyspnea is due to some added cardiac or pulmonary complication.

The pulse immediately after injury may be slow. Tachycardia with more or less irregularity is the rule. Immediate tachycardia is irritative or reflex in origin, has not the serious import of tachycardia due to cardiac injury, hemorrhage, tension pneumothorax or infection. This is to be remembered if radical measures are urgently indicated. An immediate rapid pulse per se should not cause delay.

Hemoptysis is a positive sign of lung injury. It is immediate and nearly always present in contusions of the lung. When early, profuse and continuous, it is indicative of a wound in or close to the main or a large bronchus. Hemoptysis is frequently delayed in penetrating and traversing wounds of the lung. This type of wound is nearly always accompanied by some degree of pneumothorax which may be and often is sufficient to promptly compress the wounded area of the lung and temporarily block its bronchotracheal communication. Hemoptysis may be absent in serious lung injuries accompanied by pneumothorax.

Traumatic pneumothorax may arise from an open wound in the parietal wall, in the lung or large bronchi. That from a small and promptly sealed external wound is slight and transient. It may be slow in developing and remain unrecognized for days. Pneumothorax is immediate and massive if the opening in the wall or bronchus is large and remains patent. It progressively increases in valvular types

of openings either in the lung or thoracic wall. A tension pneumo-
thorax may rapidly develop to a grave or fatal degree. (Coughing,
straining, restlessness, excitement, forced breathing aggravate the
damage of the initial trauma and increase the degree of tension
within the pleural space.)

In open pneumothorax (internal or external) air is sucked or
forced through the wound into the pleural cavity during respiration.
The suction acts with equal force on wounds and open vessels within
the lung, favors continuance of hemorrhage and tends to dislodge
clots sealing the vessels, leading to recurrent hemorrhage. The nega-
tive intrapleural pressure is also a factor in the development of
transudates and the increase of exudates in the pleura.

Therapeutic pneumothorax eliminates the effect of suction, com-
presses wounds, vessels and bronchi, prevents hemorrhage and rests
the lung, giving assurance of healing. In the absence of manometri-
cally controlled air induction, the clinical signs of absence of respira-
tory murmurs on the injured side must be secured and maintained to
assure an efficient degree of compression.

Hemothorax is an infallible sign of damage to the lung and
pleura. Hemorrhage from rupture or laceration of the great vessels
in the mediastinum or root of the lung is immediate, fulminating and
massive. Early, slowly progressive or recurrent hemorrhage is
increased by excursions of the lung. Hemorrhage from the periphery
of the lung is moderate or small, may take hours or days before it
becomes demonstrable. Existing pleuritic adhesions tethering the
lung to the chest wall increase the volume and duration of hemo-
thorax and interfere with the control of bleeding by preventing
collapse and compression of the lung.

The volume of blood in a hemopneumothorax is in inverse ratio
to the volume of air present. The amount and rapidity of bleeding
are determined by the character of the traumatizing force, the
caliber and location of the bleeding vessels, their relation to the free
pleura, the motility of the site of the bleeding vessels, the influence
thereon of the pumping action of the expanding and contracting
diaphragm, chest wall and lung during respiration, and the time
required for establishing a controlling degree of intrapleural positive
pressure. The prompt induction of pneumothorax to control intra-
pleural bleeding is founded on sound physiologic and mechanical
principles.

The blood in hemothorax clots slowly, partially or not at all. The
causes are imperfectly understood. Clotting time is influenced by the
rapidity of the bleeding. The slower the bleeding and the smaller

the volume of blood the less likely is clotting to occur. This interval favors precipitation of fibrin, permits tissue and pleural reaction to occur which modifies the blood and inhibits or prevents clotting. In massive acute hemothorax there is little or no time for pleural reaction and precipitation of fibrin. The blood clots and is absorbed slowly.

Blood in the pleural cavity changes in a short time. This time element is the basis for the diagnostic aspiration test for massive and recurrent bleeding wherein the blood nearly always coagulates.[6] It also explains why the blood is not altered in massive hemothorax and can safely be used early for autotransfusion.

The blood in a hemothorax may be rapidly absorbed at least in part. This partial absorption permits early reëxpansion of the lung, favors reopening of wounds and recurrence of hemorrhage. Early reëxpansion of the lung must be avoided. The first blood entering the pleural cavity in the formation of a hemothorax is sterile. That entering later may be contaminated with bacteria from the lung and bronchi. The presence of fluid or clotted blood in the pleural cavity is an ideal medium for bacterial growth. A moderate or small hemothorax is ample to sustain and propagate any contaminating bacteria. The possible contamination of continuing or recurrent hemorrhage emphasizes the advisability of aspirating all the blood and prevent recurrent bleeding by therapeutic pneumothorax.

Blood within the pleural cavity and the deposition of fibrin on the pleural surface inhibits whatever resistance to infection the pleura may possess. It excites a reaction pleuritis with the formation of plastic exudate and intractable adhesions. These must be prevented.

Pneumothorax will promptly control or stop bleeding from the mid-lung field or periphery. That from the vessels near or in the root of the lung is less effectively controlled. Bleeding from the parietal wall, particularly the intercostal or internal mammary vessels cannot be controlled by pneumothorax.

Fractional aspiration of blood is not so effective as complete aspiration and replacement with air. Introduction of air coincident with aspiration of the blood and fluid makes complete aspiration possible without the danger of unpleasant and occasionally serious reaction due to too sudden altered intrapleural pressure.

Fractional aspiration in some instances may be harmful. By permitting early reëxpansion and active movement of the lung, it favors continuing or recurrent bleeding and auto-infection of the pleural cavity. Fractional aspiration does not completely remove the

fertile media for bacterial growth. It does not eliminate the blood which in any amount inhibits the resistance power of the pleura. It does not prevent the deposition of plastic exudate and the formation of adhesions.

It is logical, as soon as the patient reacts from his injury, to aspirate as much of the blood from a hemothorax as possible and coincidently induce and maintain a manometrically controlled pneumothorax. In the absence of a pressure manometer the volume of the replacing air should exceed by 200 or 300 c.c. the volume of the fluid aspirated or air should be replaced until signs of respiratory murmurs in the lung disappear and the injured lung becomes silent.

Inducing and maintaining complete air replacement secures collapse of the lung, compresses wounds, closes bleeding vessels and open bronchi, the commonest source of infection, eliminates the media for bacterial growth, prevents the formation of plastic exudate and tethering adhesions,[7] reduces reaction pleuritis and inflammatory processes in the lung, rests the lung and favors healing.

Blood or fluid of any kind does compress the lung. Fluid is inelastic, non-compressible, and does not accommodate itself to the volumetric changes of the lung during respiration. Air of less than atmospheric pressure is elastic, diffusible, compressible, follows the slightest movement of the lung making for constant uniform compression and efficient immobilization. Air does not embarrass to the same degree as fluid the retractible power of the opposite lung.[7] Manometrically graded pneumothorax is ideal for controlling all factors of injuries to the lung which do not urgently demand radical measures. The earlier it is induced, the more effective.

Compression injuries of the heart are usual sequences of blunt trauma to the chest. (The position of the heart between the sternum and spine make it susceptible to injury by compression.) The symptoms may be mild and transient, moderate, lasting for hours; severe, persisting for days or months; fatal resulting in immediate or delayed death. There may be no external or superficial evidence of injury yet the cardiac damage may be permanent or fatal. Postmortem findings of cardiac compression in immediately fatal cases may be inconclusive or absent. Those in delayed or late deaths show positive evidence of coronary and myocardial pathology, ecchymosis, focal hemorrhage, pericarditis, epicarditis, myocardial fibrosis.

Symptoms may be those common to shock—syncope, pallor, an imperceptible, small, slow or rapid, intermittent, irregular pulse. Electrocardiogram indicates disturbance of conduction. The arterial blood pressure falls, the venous pressure rises, heart sounds are faint.

The symptoms are reflex in nature. The reflex may originate from the surface of the body or from the viscera and is reflected over the sympathetic and central nervous systems, affecting the heart, the peripheral, cerebral and splanchnic vessels.

In severe compression, pain in the cardiac area may be sharp and lasting. There may be a true traumatic angina pectoris with contraction of the coronary vessels, acute myocardial ischemia, cardiac embarrassment, dilatation and a cerebral anemia. If this lasts long enough, myocardial damage with all the characteristics of coronary block, fibrosis, scarring, thinning of the wall, local aneurysmal dilatation, and cardiac decompensation[8,9,10] will develop. These pathologic changes emphasize the importance of rest and obligate a guarded prognosis during the first weeks following compression of the chest with severe or persistent cardiac symptoms.

Fracture of the ribs may be single, multiple, comminuted or compound. Such a fracture can usually be recognized without moving the patient. Fractures of the sternum and cartilages, unless marked deformity is present, are difficult to diagnose even with the x-ray. The clinical signs of point tenderness (which is difficult to elicit in the upper four ribs posteriorly) and pain on pressure in a fixed area warrants the diagnosis. Complex and comminuted fracture of a rib or ribs is more dangerous since the fractured ends and bone spicules commonly cause puncture, laceration or other injury to the pleura and lung. Fracture of a single rib due to blunt trauma rarely causes hemorrhage from the intercostal vessels.

Fractured ribs may have little influence on the intrathoracic function unless accompanied by intrathoracic damage. The reverse is true in penetrating wounds especially, shot and stab wounds, in which the internal damage is always greater than the external evidence indicates. The damage to the chest wall in closed injuries is not so hazardous as that sustained by the contained viscera: contusion, rupture of the heart, contusion laceration of the lung and pleura, hemopneumothorax. Internal damage must be looked for by frequent recheck examination. The chest should not be strapped with adhesive, especially in upper thoracic wall injuries. Strapping may aggravate the damage, and interferes with reëxamination and observation of the course of intrathoracic injury.

Emphysema may be subcutaneous, subpleural or mediastinal; local, spreading or generalized.

Localized subcutaneous emphysema occurs in valvular or oblique wounds in the extrathoracic structures, the air being sucked in and spread by muscular contractions and movement.

Spreading emphysema may develop in open or closed wounds communicating with the pleural space and punctured and lacerated wounds of the lung. The air from within the chest is forced into the subcutaneous tissues more rapidly than it can escape through the opening in the skin. It is most frequent and more generalized in wounds of the lung and bronchi which are held by adhesions in open communication with the subcutaneous tissues, the overlying skin being intact. The larger and more direct this opening, the more extensive and rapid the development of emphysema. It usually develops promptly, spreads rapidly throughout the loose cellular tissue of the neck, face, trunk and extremities.

Coughing, straining, labored breathing, and muscular effort raise the intrathoracic pressure, increase the escape, accumulation and spread of air in the cellular tissues.

Subcutaneous emphysema is not dangerous, but the underlying injuries may be very serious. If escape of air cannot be controlled at the opening in the lung or chest wall, needle the pleura to determine the presence and degree of tension pneumothorax. It may be possible to reduce and control the intrapleural tension and stop the subcutaneous escape of air.

Mediastinal emphysema is most frequent in severe compression of the chest with no open external wound. It may complicate wounds at the base of the neck, operations for substernal and intrathoracic goiter, retraction of the bronchial stump after lobectomy or pneumonectomy. Mediastinal emphysema develops and spreads rapidly and may attain a high degree of tension.

The symptoms depend on the rate, amount and pressure of the air accumulating in the mediastinum. There is progressive dyspnea, cyanosis, bilateral venous congestion in the lungs, dilatation of veins in the neck and upper chest, pain dysphagia, extra pericardial pneumatic tamponade, circulatory failure and death. Physical signs first appear in the jugulum and base of the neck as a soft crackling swelling which rapidly extends up the neck to the face and head, into the axillae, down the arms, chest and abdomen. There is tympany over the entire sternum, breath sounds are reduced. Heart dullness is obliterated, the heart sounds become progressively fainter. There are crackling râles synchronous with the heart beat. Small amounts of air may be overlooked. X-ray films show mottled areas with vertical columns of decreased mediastinal density. The large vessels are well differentiated.[11]

The treatment is transverse incision over the jugulum, cutting the attachment of the strap muscles to the sternum, and the insti-

tution of cup or suction drainage. If the condition is not promptly relieved the mediastinum is opened parasternally or the sternum split.

GENERAL OUTLINE OF TREATMENT

Closed Wounds. Treat shock, prevent or control hemorrhage, cardiac and respiratory embarrassment. Morphia is given in sufficient dose to assuage pain, to slow and quiet respiration, to reduce anxiety and restlessness. Small doses repeated at short intervals are better than large doses at long intervals. Oxygen inhalation is necessary for dyspnea, and graded transfusions to restore and maintain the circulating media. Blood is better than solutions of glucose or acacia in such transfusions.

In a careful general physical examination, note the gross intrathoracic and circulatory status, moving the patient as little as possible. Check the progress by reëxamination until the patient begins to improve. For immobilizing the chest wall apply a snug binder rather than adhesive straps, and thereby avoid moving and rolling the patient, and obscuring physical signs.

Reduce tension pneumothorax and maintain a comfortable degree of compression by repeated needle aspiration or a small indwelling trocar, with a connected water valve trapped tube.

In hemothorax, carry out early aspiration of all the blood possible, using it for direct autotransfusion if hemorrhage is massive.[12,13] Replace the volume of blood aspirated with a slightly greater volume (200 to 300 c.c.) of air. Check the intrapleural pressure manometrically.

Fluoroscopy and roentgenography are necessary as soon as possible after reaction from shock.

Direct surgical attack for intrathoracic cardiac and subdiaphragmatic damage is at times urgently demanded. To cope successfully with these, especially in injuries of the closed type, taxes the judgment and ingenuity of the most versatile surgeon. Surgical attack for injuries to the heart, the great vessels at the base, in or near the root of the lung or in the mediastinum is imperative and most formidable. Open radical operations are reserved for progressive uncontrollable hemorrhage, mechanical respiratory and cardiac embarrassment, mediastinal emphysema, wide open defects in the thoracic wall, laceration of the lung, puncture of the heart, laceration rupture of the diaphragm and subdiaphragmatic viscera.

General anesthesia is used intratracheally or with a positive pressure mask.

For upper chest damage the approach is through the periosteal bed of the resected fourth rib; for mid or lower thoracic exploration, through the periosteal bed of the resected seventh rib. Additional ribs are cut anteriorly or posteriorly to enlarge the opening if required for thorough inspection and workable space. A pliable curved or angled rubber guarded clamp is applied to or a rubber tourniquet applied around the root of the lung to control bleeding. Foaming areas or points indicate the site of wounds in the lung. Bleeding is arrested by ligating vessels separately or by mass mattress suture. Damaged tissue is excised, foreign bodies removed, the lung sutured and inflated to test the sutures and determine the presence of any bleeding vessels. The surgeon explores for a rent in the diaphragm, removes all clots and blood, flushes the pleura, and sutures the periosteal incision. Tension on the suture line is relaxed by coapting the rib above and below with approximating sutures, closing the wound in layers as airtight as possible and injecting air into the pleural cavity to a mildly compressing degree. Postoperative repeated aspiration of accumulated fluid is necessary, accompanied by air replacement. If imperative later, a dependent counterdrain may be inserted by the closed method.

Open Wounds. Large defects in the thoracic wall must be immediately occluded and sealed. One may use the flat of the hand, a folded wet towel or cloth, cellophane, or rubber dam or one may insert a large moist soft gauze tampon, or pull the deflated lung into and block the opening. This temporarily controls the sudden massive collapse, steadies the mediastinum, and relieves cardiac and respiratory embarrassment. Immediate operation is then done under positive pressure anesthesia. The wounds and pleura are cleansed and closed and the lung compressed. The wound is debrided, fragments of comminuted ribs removed as well as all non-viable tissue and tags, all clots and blood. Bleeding vessels in the wall and lung are ligated, as are open bronchi. The lung is sutured and the pleural cavity flushed. The wound is closed, if possible, airtight. The lung is rested, sealed and healed by a mildly compressing degree of pneumothorax. If fluid accumulates, one must aspirate repeatedly and if and when pus develops, drainage by the closed method is done.

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DISCUSSION

JAMES M. WINFIELD (Detroit): It was a great pleasure to have heard Dr. Hegner's excellent paper.

The indications for aspiration of blood and replacement by air in those cases where there has been profuse intrapleural bleeding have concerned us greatly in the past year. We therefore chose a group of forty selected wounds of the chest which were penetrating in type. There were no complicating injuries of any moment. So far as we know, only the lung was involved, the great vessels and the heart escaping injury.

We divided these cases into those that had hemothorax, hemopneumothorax, and pneumothorax, and also as to whether they had a slight, moderate, or severe clinical reaction. The average number of days and the spread of days of hospitalization were listed for each type of case. When hemothorax alone was present, and a slight clinical reaction, the average number of days in the hospital was six, with a spread of four to eight days. When there was moderate clinical reaction, the average number of days in the hospital was eight with a spread of eight to ten days, and when there was a severe clinical reaction, the average number of days in the hospital was eighteen, with a spread of thirteen to thirty-three days. When hemopneumothorax was present, those cases with slight clinical reaction had an average number of days in the hospital of seven (three to fourteen). With moderate clinical reaction, the average number of days in the hospital was seven, with a spread of six to fifteen days; with severe clinical reaction, the average number of days in the hospital was nineteen, with a spread of fourteen to twenty-eight.

When pneumothorax alone was present, the patients exhibited only slight to moderate clinical reactions, and the average days in the hospital were five or six with a spread of three to eight. In comparing the three types of cases, it will be found that those patients exhibiting hemothorax and hemopneumothorax practically parallel each other in reactions and in

the number of days that they remain in the hospital. Those patients having pneumothorax alone had markedly less clinical reaction and their stay in the hospital was much shorter than those of the other two groups. It therefore seems that the important element as far as determining the reaction and morbidity is concerned, is the presence of blood in the pleural cavity. Also it seems evident that the greater the amount of blood, the more severe the reaction and the longer the stay in the hospital.

In only three of these cases was blood aspirated from the pleural cavity, with air replacement. One of these was a tension pneumothorax, and two had a rapidly increasing amount of blood with severe respiratory difficulty.

Possibly it would have been wise if we had aspirated the blood from the pleural cavity in a larger number of these cases, as this procedure might have lessened the patient's clinical reaction and shortened the patient's morbidity and stay in the hospital. It would be extremely interesting to me to know whether Dr. Hegner has any information regarding this particular point, that is, whether he has a more or less parallel series of cases and whether he found that the procedure of aspiration and replacement by air gave better results.

HUGH H. TROUT (Roanoke, Virginia): Dr. Hegner referred to chest injuries in the late War. He stressed that there was a difference in the types of hospitals in which these patients received attention. I would like to add there was also a véry marked difference in the type of patient we treated then and the patient we now treat.

During the World War Dr. Yates of Milwaukee and his associates operated on a great many more puncture wounds of the chest than the rest of us did. The rest of the group treated them "conservatively." Dr. Yates' immediate mortality was higher than our mortality. However, in going over those cases for a period of ten years after the War his mortality was lower than ours, for a great many of our patients died later from lung abscesses, general sepsis, etc.

I know of nothing that is more distressing to both the patient and the surgeon than to have the patient plastered up with a lot of adhesive straps. We bind our chest cases with a many tailed bandage which can be easily removed or tightened as indicated.

Immediately upon admission to the hospital, if it is possible to take an x-ray picture with a portable machine without disturbing the patient too much, this is done, not because the information obtained is of any great value, but to have the plate as a check for future x-ray studies. With the majority of cases no more can be done with the x-ray for a few days until the patient is able to stand in an erect position. Of course, the fluoroscopic examination of the chest is of more importance than any number of x-ray plates.

The complete and immediate removal of blood from within the pleural cavity with replacement of air is, I think, probably the greatest advance

in the conservative treatment of puncture wounds of the chest. It is a great pity that the manometer is not used more frequently, for it not only makes the replacement of air more scientific, but gives far greater comfort to the patient. Various fluids and antiseptics have been tried over the years, but air is far preferable for the replacement medium. If you do not have a manometer, 200 or 300 c.c. more air than removed fluid should be injected. We have never used the blood removed for autotransfusion, for even in early cases I think there is always too much danger of clots, infections, etc. We believe it is important to use small and frequently repeated doses of morphia rather than few large doses.

If anesthesia is necessary it should be given by the intratracheal measure or at least by a positive pressure apparatus.

In a few cases we have used sulfanilamide both by mouth and directly on the wound. I do not know whether it has been of any value or not, but in any case the administration of this drug should be checked by blood examination and close clinical observation. Our general rules are: (1) to operate on all sucking wounds; (2) to explore all puncture wounds near the sternal border; (3) to operate in all cases where the lung presents itself into the wound; (4) to operate where the diaphragm might be injured; (5) to intervene where there is a tense pneumothorax or (6) where there is marked subcutaneous emphysema.

Of course, very frequently, in injuries of the chest, consideration will have to be given to the treatment of injuries in other parts of the body. Recently we have seen a few cases of thoracic hernia coming through the chest wall. Some of these required operation. As a rule, the opening can be closed by osteoplastic flaps, though in two cases we have been forced to employ fascia taken from the fascia lata.

I want to again express my personal appreciation to Dr. Hegner for this well considered and timely paper.

CASPER HEGNER (closing): The wounds of the chest that I tried to emphasize were not stab wounds or gunshot wounds. We know, of course, that infection in those is not nearly so great as where we have tissue trauma of the chest wall or of the lung. The amount of hemorrhage in the lung and in the pleura, as I said, is important, and it is a source of irritation. It does not compress as well as air. Therefore, as soon as the patient recovers from shock, I believe as much blood as possible should be aspirated and this should be replaced immediately by air. This shortens the reaction time; shortens the stay in the hospital; gives better visualization in the x-ray. In addition, you can control the initial hemorrhage, you can control recurrent hemorrhage and you can prevent secondary hemorrhage as well as infection.

THE MISUSE OF SKELETAL TRACTION*

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SKELETAL traction, long considered feasible in the minds of surgeons, had to await the introduction of aseptic surgery for its safe and fullest application. As early as 1900 Heineke[1] described a method of applying tongs to the calcaneus. In 1903 Codivilla[2] introduced the use of the nail or pin, a method which in 1907 was elaborated and popularized by Steinmann.[3] In 1912 Klapp[4] improvised a method of direct traction on the calcaneus by the use of flexible wire which was threaded through the plantar surface of the heel in order to avoid pressure on soft tissues. During the world war Herzberg[5] improved this method when he devised a means of keeping flexible wire taut. All of these forms of skeletal traction, because of the dangers associated with their use, were not generally accepted until 1927 when Kirschner[6] described a simplified means of using stainless steel wire of 1 mm. diameter as a drill in itself.

Since that time there has been a growing wave of enthusiasm for the use of all types of skeletal traction. In the wake of this enthusiasm we find a number of complications, which have led to criticism of a method which is useful and sound when properly applied. The medical literature of the last ten years is filled with suggestions for the application of skeletal traction to the treatment of almost every known fracture. Some of these have considerable merit, but are often unwarranted substitutes for older, established methods. Some surgeons have lost sight of the fact that every wire, tong or nail inserted into bone constitutes virtually a compound injury to that bone, and carries with it the hazards associated with that type of injury. Enthusiasts have sponsored the use of multiple pins and, in certain fractures, the insertion of pins directly through the line of fracture. These methods, in the hands of experts, may lead to gratifying results, whereas their general use may result in nothing but disappointment.

The rules governing the application of skeletal traction as well as the indications for its use have been set down by many competent authors and will therefore not be repeated here except to

mention that it is our conviction that the misuse of the method rather than the method itself leads to most of the difficulties associated with it.
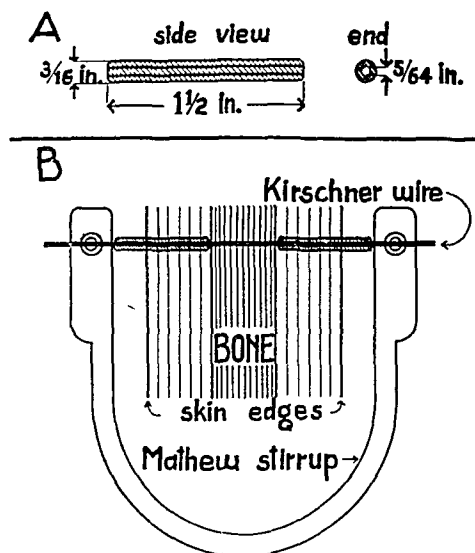
FIG. 1. Metal sleeve to prevent longitudinal axis motion of skeletal pins. A, dimensions of sleeve. B, sleeves in place. (Courtesy of Dr. Bret Smart.)

FIG. 2. Kirschner wire through lower end of femur with metal sleeves being placed. (Courtesy of Dr. Bret Smart.)

INFECTION

The greatest hazard associated with the introduction of metal into bone through the soft parts is that of infection. Infection may occur in the skin, in the underlying soft tissues or in the bone itself.

As a general rule it starts in the skin and extends by continuity into the bone. This, however, is not always so. Bacteria may be carried directly into the bone by the introduction of the metal

FIG. 3. Metal sleeves in place over Kirschner wire through ulna. (Courtesy of Dr. Bret Smart.)
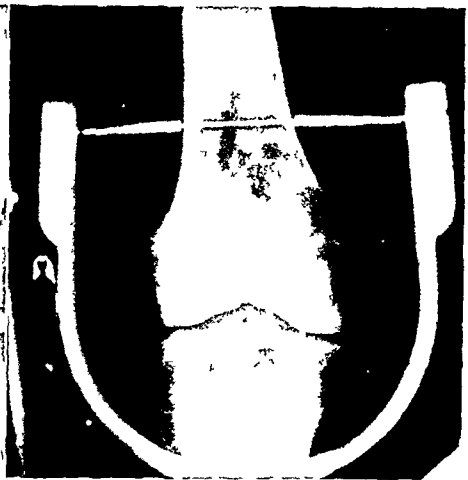
FIG. 4. Roentgenogram illustrating metal sleeves in place. (Courtesy of Dr. Bret Smart.)

agent through improperly prepared skin. Many surgeons take no other precaution than to swab the skin at the point of introduction

A   B   C
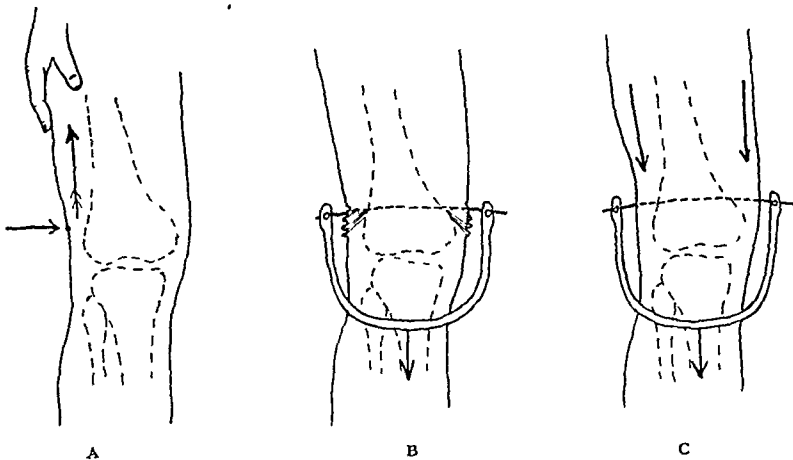
FIG. 5. Diagram: A, direction in which skin should be pulled before inserting traction pin. B, area of skin necrosis if skin is not properly treated. C, same when properly treated.

with a disinfecting agent. This is often the first link in the chain of misfortune that will follow its general adoption. Too frequently the insertion of a pin is treated as a minor surgical procedure with the

FIG. 6. Arrows indicate ring sequestra of cortical bone following removal of traction pin.

FIG. 7. Arrow indicates area of pressure necrosis in bone following skeletal traction. Note that wire has pulled out of bone into the soft tissues.

A   B   C
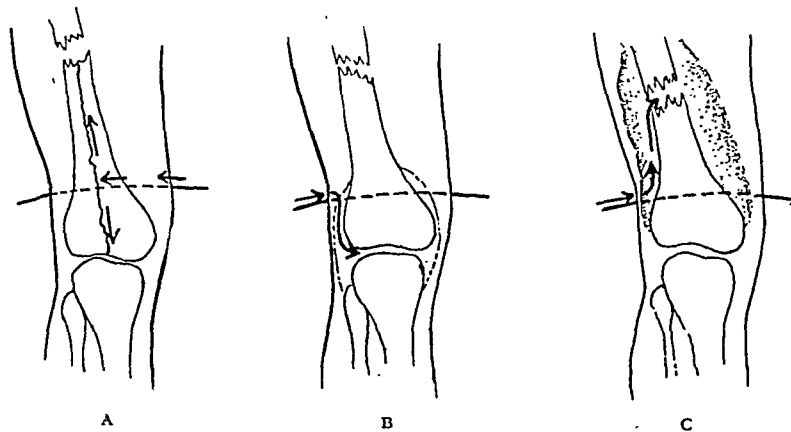
FIG. 8. Skeletal pin inserted through A, linear fracture communicating with the major fracture site and joint; B, a joint capsule; C, a hematoma which originates at the fracture site. Arrows indicate the course taken by invading bacteria.

patient in bed. The propriety of meticulous surgical technique cannot be over-emphasized.

Once the pin is inserted it must be fixed in place. Subsequent

FIG. 9. A, skeletal pin through a linear fracture in os calcis. B, resultant osteomyelitis of os calcis.

FIG. 10. Gangrene of thumb following skeletal traction through terminal phalanx.

motion of the pin, particularly along the axis on which it was driven, will inevitably lead to infection. The consequences of this infection may or may not be great but it must be avoided at all costs. Motion in this direction is not apt to occur immediately after the pin has been inserted, because at first it is firmly embedded in the bone. Later, as traction is applied, aseptic pressure necrosis takes place in the bone, which allows the pin to become loose. Movement of the patient thereafter displaces the pin in its longitudinal axis so that the contaminated portion protruding from the skin slides into the subcutaneous tissues, carrying bacteria with it. This misfortune, as will be mentioned later, may lead to dire results.

Recognition of this danger has led to various means of prevention. Many surgeons insist upon using Steinmann pins rather than wire for this reason alone. It is true that Steinmann pins become fixed more firmly in bone, but they too loosen with time. When they

do they are more apt to cause trouble than finer wire because their initial damage to the bone is greater. Some operators depend upon material such as cork, rubber sponges or wrappings of gauze inserted

FIG. 11. A, skeletal pin through os calcis at improper angle. B, sliding of pin with resultant pressure on soft tissues.

between the skin and the spreader to prevent longitudinal axis motion of the pin. All of these agents are, to a certain degree, unsatisfactory: some are too soft and allow a certain amount of motion; others are too hard, and cause pressure necrosis of the skin.

Recently we have been using the metal sleeves shown in Figures 1 to 4. These sleeves are made in varying lengths so that they fit firmly between the cortex of the bone and the spreader. After the wire is inserted into the bone the sleeves, one on each side, are forced along the course of the wire firmly against the surface of the bone through small incisions in the skin at the points of exit of the wire. The spreader or stirrup is then fixed firmly against the outer limits of the sleeve, preventing any motion in the longitudinal axis of the wire. The same motion also can be prevented by encasing the pin in plaster of Paris, provided there is no space left between the skin surface and the plaster encasement. It is a mistake to place bulky dressings over the openings in the skin under plaster. We prefer

to use no dressing at all. The dried-up secretions around the junction of the pin and the skin surface will prevent the invasion of the soft tissues with bacteria.

A   B

FIG. 12. A, skeletal pin placed at improper angle. B, roentgenogram of same case.

Proper asepsis and fixation of the pin alone will not necessarily prevent infection. Once traction is applied the pull exerts itself, not only on the bone but also on the soft tissue in contact with the traction pin. Unless these tissues are freely movable, traction upon them will cause pressure necrosis and subsequent secondary infection. The amount of necrosis depends upon the resistance of the soft tissues to the force applied, and may be prevented by pulling them in the opposite direction to the expected line of pull at the time of insertion of the traction pin. (Fig. 5.) Allowance also should be made for a certain amount of bowing in the metal agent used for traction. The soft tissues in the region of a pin used for counter-traction must be similarly treated, keeping in mind the anticipated direction of pull.

Infection starting in the soft tissues may remain localized in the skin or subcutaneous tissues or it may extend along the course of the pin into the transfixed bone. Once the infection has reached the bone it may confine itself to the osseous tissue immediately in

contact with the pin or may spread to the medulla with a consequent widespread osteomyelitis.

If the infection is mild and the pin remains fixed in position the

FIG. 13. Bone split by insertion of skeletal pin.

final outcome is apt to be a so-called "ring sequestration" of the cortical bone, as seen in Figure 6. These sequestra are often the cause of persistent draining sinuses. On the other hand, motion of the pin in the presence of infection will lead to more or less extensive bone damage. Constant traction under these conditions may result in migration of the pin out of the bone into the surrounding soft tissues or through damaged bone into a nearby joint or epiphyseal area. (Fig. 7.) Accidents of this type, in addition to causing prolonged suppuration, may result in disturbances of bone growth, destruction of joints and even loss of limb and life.

Not infrequently hematomata resulting from local trauma or spreading from the site of fracture are unwittingly traversed by the traction pin. (Fig. 8.) It is well known that hematomata offer the best possible conditions for the growth of bacteria, regardless of their number or virulence. Infection originating in this fashion will

extend, not only to the soft parts invaded by the hematoma but by direct continuity to the fracture site. As Kellogg Speed[7] has pointed out, "The surgeon must recall that the hematoma may

FIG. 14. Distraction in a fractured femur.

extend from one end of the limb to the other and even beyond some of the larger joints in severe injuries." Not infrequently a joint capsule or a bursa communicating with it are penetrated. Joints and bursae, as well as hematomata, offer excellent culture media for the growth of bacteria. Similarly, and in our experience more frequently, unrecognized linear fractures extending in the longitudinal axis of the bone from a major fracture site are traversed. (Fig. 8.)

Infection originating in the skin may extend along the course of the pin into the linear fracture line and then find its way to the major fracture. (Fig. 9.) In the past year we have seen empyema of the knee joint in two patients, both with fractures of the femur, treated with skeletal traction. In each of these patients there was an unrecognized linear fracture extending from a transverse supracondylar fracture into the knee joint. Both patients had a Kirschner wire driven through the lower end of the femur at some distance from the transverse fracture but directly through the linear fracture which communicated in each case with the knee joint.

Injuries to nerves and blood vessels either from penetration by the metal agent at the time of introduction or by subsequent pressure or infection may lead to permanent damage in the limb.

FIG. 15. Distraction in tibia and fibula.

Figure 10 shows gangrene of the thumb which followed the penetration of blood vessels by a metal agent inserted through the terminal phalanx.

ATTENTION TO DETAILS

Improper attention to details often leads to disaster in the use of skeletal traction. As a general rule, it may be stated that pins should pass through the bone at a right angle to the anticipated line of pull. The surgeon must be fully cognizant of what that line of pull should be before inserting the pin. Not infrequently a pin is inserted with the limb lying in the uncorrected position so that after the correct alignment is obtained the pin is found to pass at an unsatisfactory angle through the bone. This not only leads to inefficient traction but causes the pin to slide through the bone on its longitudinal axis. This, in turn may lead to infection or pressure necrosis of the soft tissues as seen in Figure 11. This is most apt to occur in injuries about the ankle joint where the os calcis is used as the point of skeletal attachment. Inversion or eversion of the foot tends to

FIG. 16. A and B, rotatory displacement in tibia maintained by skeletal
fixation. C, nonunion after six months. C, nonunion after eight
months. Note that fibula has united.

mislead the surgeon as to the proper angle at which the traction pin should be placed. (Fig. 12.)

Unintelligent selection of the type of metal agent to be used as

FIG. 17. Displacement of tibia maintained by fibula. Arrows indicate ring sequestra and linear fractures.

well as poor condition of the apparatus may result in trouble. Too large or too dull a pin may split the bone into which it is driven. (Fig. 13.) Pins which have been boiled too frequently lose their tensile strength and will bend under heavy traction. Bending may make it extremely difficult to remove the pin without undue damage to the bone or may cause splitting. We recall one patient in whom the femur was split through its entire length as the result of bending of a Steinmann pin under heavy traction.

The criteria for selection of the metal agent to be used for traction in various types of fractures, although of great importance, cannot be discussed here. Usually the danger of damage to the bone and of infection is directly proportional to the size of the metal agent used.

Likewise a discussion of metal tongs has been omitted. They are a useful device, particularly for skeletal traction on the skull in fractures of the cervical spine. Elsewhere they are extremely danger-
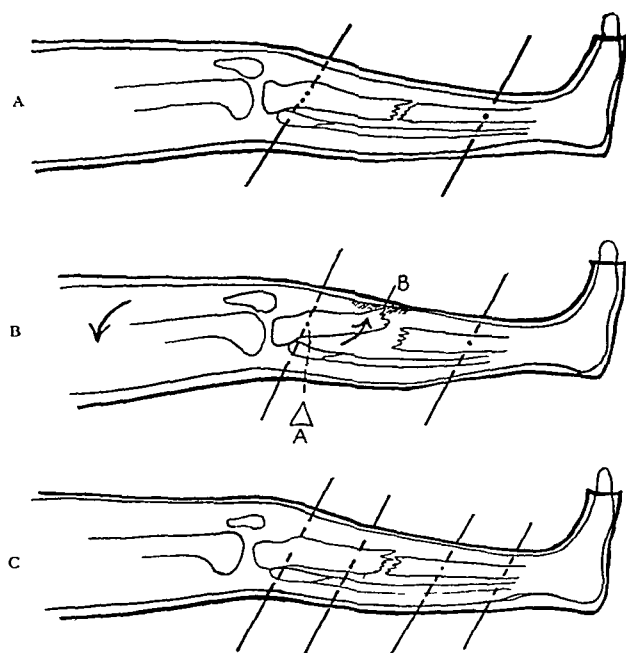
FIG. 18. A, skeletal pins above and below fracture site. B, rotation of upper fragment at point A as a fulcrum causing pressure necrosis at point B. C, multiple pins to prevent such rotation. This method is condemned by the author.

ous, even in the hands of experts, because of their tendency to slip, causing extensive and severe damage to the soft tissues.

DISTRACTION

Aside from infection, probably the greatest danger associated with skeletal traction is that of distraction, leading to delayed union or nonunion. Skeletal traction has provided the surgeon with an exceedingly efficient means of direct pull on the skeleton which, because of its inherent power, may prove disastrous. Too much weight or the use of an appropriate weight over too long a period of time will not only distract fragments but will stretch the musculature to the point that it loses its power to contract. (Figs. 14 and 15.) Prolonged distraction may be as difficult to correct as prolonged shortening. Distraction, if recognized early, can usually be corrected by decreasing the amount of pull, if during the period of

distraction soft parts or periosteum have not become interposed between the bone ends.

Frequent x-ray examination is of value to check the amount of
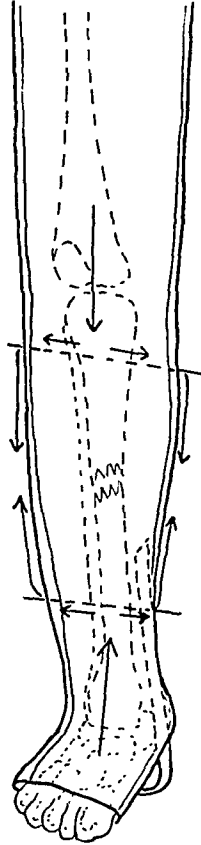
FIG. 19. Lines of weight bearing when two pins are incorporated in a walking cast. Note that there is no weight bearing on the fracture site.

pull but should not be the only means of control. Roentgenograms, unless taken in exactly the right plane, give an erroneous picture of conditions as they actually exist. Excellent reduction and alignment reported from the x-ray laboratory mean very little unless the clinical findings corroborate this statement. Bony crepitus felt at the site of fracture assures one that there is no interposition of soft parts and that the bone ends are in contact far more certainly than the x-ray picture.

A multitude of methods for the application of skeletal traction with single or multiple sites of skeletal attachment are now in general use. Most of the methods using a single point of skeletal fixation with continuous traction and suspension, although they present the hazards already mentioned, are comparatively safe, and in the treatment of certain fractures indispensable. The addition of secondary points of skeletal fixation to permit countertraction or pull and the use of distraction apparatus greatly increase the hazards. It would not be fair to condemn these modifications when exercised by experts. Nevertheless, a word of warning to the inexperienced is in order. In theory it is simple to pull, twist or angulate the fragments of a fracture back into place by mechanical means, using two or more points of skeletal attachment. In practice it is, unfortunately, another matter. It would be simple in most cases, were it not for the interference presented by the damaged soft tissues. The distraction force necessary to correct displacement in many cases is so great that transfixion pins bend or become displaced, or the soft tissues themselves tear, causing more extensive hemorrhage and swelling. The circulation in a badly swollen limb may become completely obliterated by long-axis traction in a distraction apparatus, particularly when tension is applied to a joint. The efficiency of skeletal traction when applied suddenly through a joint may be so reduced that it is often necessary to overextend the soft tissues in order to obtain reduction. Flexion of the fingers or toes, blanching or cyanosis of the skin, while traction is in force through the ankle or wrist joint, should warn one that the distraction force is too great. Traction under these conditions, if maintained, may result in a permanent palsy, due to ischemia. It should be remembered that muscle spasm, particularly when associated with hemorrhage or edema in the soft parts must be overcome by prolonged steady traction, rather than by a sudden powerful pull. Often shortening is due not only to muscle spasm, but to shortening of the musculature because of hemorrhage and edema in the muscle fibers themselves. This type of shortening cannot be overcome by a sudden traction or distraction force without severe damage to the muscle tissue. The immediate damage caused by overextension may lead to permanent fibrosis and contracture.

The rigid fixation provided by setting transfixion pins in plaster or in a splint after reduction has been obtained in a distraction apparatus, is of considerable advantage in maintaining reduction in certain types of fracture. Also it is hazardous. The greatest

caution must be exercised in obtaining proper reduction if this means of fixation is to be employed. The reduced fragments remain absolutely fixed in the position in which they are placed and muscle balance no longer aids in the reduction. The slightest amount of distraction between the fragments remains constant during the period of fixation and may lead to delayed union or to nonunion. Rigid fixation in spiral or oblique fractures, even when proper length has been obtained, often maintains enough separation caused by rotation to delay healing. (Fig. 16.)

In the treatment of fractures of both bones of the leg or forearm by fixation between two or more transfixion pins there is great danger of maintaining a small amount of distraction at the fracture site in one bone while the fragments of the other remain in contact. Union between the fragments in one bone will then maintain distraction in the other. This is a frequent cause of delayed union and nonunion. (Fig. 17.)

Although absolute fixation to prevent shortening or rotation can be maintained by fixation with one pin above and one below the fracture site, angulation in the anterior-posterior plane cannot be avoided unless more than two pins are used. This is a common fault of the two-pin fixation method of treating fractures of the shaft of the tibia. Excellent reduction may be accomplished and the leg placed in a long leg cast with the pins incorporated in the plaster, only to find that motion of the body leads to rocking of the upper fragment with the upper pin acting as a fulcrum. (Fig. 18.) The resulting angulation at the fracture site may lead to pressure necrosis of the skin over the tibia and to compounding of the fracture. (Fig. 18.) The use of multiple pins to prevent this rocking motion is dangerous and unwarranted.

During the past few years the ambulatory treatment of fractures has grown in favor. Many fractures, particularly those of the lower extremities, which ordinarily could not be treated in this category for fear of displacement, are now allowed early weight bearing on transfixion pins incorporated in walking casts. Notwithstanding the admitted advantages of early weight bearing this practice should be discouraged. Walking in a cast with a transfixion pin above and one below the fracture site does not allow weight bearing at the site of fracture, which is of advantage. (Fig. 19.) Again, ambulatory treatment under these conditions tends to maintain any existing distraction and to favor development of the rocking motion previously described. Weight bearing on transfixion pins is also liable to loosen the pins and encourage infection. Finally, patients under

treatment with skeletal traction must be under constant supervision and definite control which this type of ambulatory management will not allow.

SUMMARY

The misuse of skeletal traction rather than the application of the method itself leads to most of the difficulties associated with it.

The greatest danger of the method is infection, which may result from improper surgical technique at the time of application or from subsequent motion of the metal agent used for traction. A method of preventing longitudinal axis motion of skeletal pins is described.

The complications of infection are most severe when a hematoma, joint or linear fracture communicating with a major fracture are traversed by a traction pin.

Improperly placed pins may result in inefficient traction, pressure necrosis of soft tissues or severe damage to the bone or neighboring epiphyseal areas and joints.

The use of metal tongs is condemned, except for traction on the skull.

The problem of distraction resulting in delayed union and nonunion is discussed.

The hazards associated with the use of multiple points of skeletal fixation and distraction types of apparatus are emphasized.

The ambulatory management of patients treated by the incorporation of skeletal pins in plaster is considered dangerous, particularly when weight bearing on the skeletal pins is allowed.

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DISCUSSION

GUSTAV F. BERG (Pittsburgh): I think that skeletal traction by the use of the Kirschner wire, the Steinmann pin or the ice tong is going to lead us into an occasional difficulty. To any individual who intends to treat fractures, I should like to make a few recommendations.

Let us discuss a fracture of the femur. In Dr. Kellogg Speed's book on fractures, he describes the anatomic location of the fracture, laying particular stress on the muscles attached to the different regions of the femur. The fracture of the upper third always assumes a characteristic position, caused by the pull of the attached muscles. If a fracture is seen early and receives proper attention, it can be reduced, and if the proper type of fixation apparatus is applied, it can be held in its proper position.

In case you desire to use skeletal traction in a fracture of the femoral shaft, there is a scientific way to go about it. The x-ray findings should be studied, a diagram made of the fractured shaft of the femur, and the angles in which the wires are to be inserted should be considered, in order that when proper traction is applied, the traction is in such a direction that it will both correct and prevent any recurring deformity, and not alter the proper angle of the articular surfaces of the joints. It is important to remember that Kirschner wires have a tendency to bend, and any appliance used to control the Kirschner wire must have attachments to prevent this bending.

I feel that in many cases, the incorporation of the Kirschner wire or the Steinmann pin in a plaster cast is a mistake if the fixation material is a circular plaster cast which prevents early motion of the adjoining joints.

In looking back over past experiences, what have been the anatomic structures which were responsible for the disability following fractures? Is it not a fact that the contracted structures surrounding the joints, the myofascitis and the fibrositis are the result of long continued disuse and that such factors must be eliminated? In dealing with a long spiral fracture, what usually happens? A piece of bone pushes its way through the soft structures and it is at times impossible to free the soft structures by the use of skeletal traction. At the removal of the traction the fragments are still displaced and the interposing soft structures cause nonunion. I am of the opinion that following the anatomic approaches as laid down by Dr. Henry, a skillfully performed open reduction, using the nofinger touch technique, performed by a competent surgeon, will give the best end result.

R. ARNOLD GRISWOLD (Louisville): This is a most timely paper of Dr. Mathewson's. Those of us who are enthusiastic about the use of skeletal traction fully realize, I think, the dangers and the pitfalls of this method. Skeletal traction with its logical sequences of countertraction and skeletal fixation in plaster are operative surgical methods, and they have all the dangers of abuse and misuse that other operative surgical measures have. They must be carried out with due regard for their indications and

contraindications, and with meticulous attention to the details of the technique.

Obviously, such operative methods are not indicated in fractures which can be handled by external manipulative reduction and external fixation. One sees double Steinmann pins or double Kirschner wires used in cases which should have been treated by simply manipulative reduction and plaster cast. More radical methods should be reserved for those cases in which such reduction is impossible, as for example, the compound fractures in which we have extensive comminution which prevents locking of fragments and in which we desire some method of adequately treating and observing the wound without sacrificing position. Also, in those simple fractures in which position cannot be maintained except by open operative methods.

From the standpoint of infection, I think the likelihood of bone infection is less in the hands of the average man when skeletal fixation is used, inserting the pins at a distance from the hematoma, that it is if that same man did an open operative reduction and internal fixation. Adherence to the details of technique implies that we consider this a major surgical operation and insert the pins with all the ritual of such a procedure. It is not a thing to be done in bed or in a farm house or on a kitchen table.

The points which Dr. Mathewson has brought out cover almost all my ideas of the details of technique, the proper insertion of the pin in relation to the line of pull, and avoiding the insertion of the pin through the hematoma of the fracture. The hematoma is much more susceptible to infection than are the bone and soft tissue at some distance from the fracture. In inserting the pins, we drive them through the bone in order to increase the amount of fixation and prevent motion. It is extremely important to drive pins, however, only through the cancellous structures at the extremities of the bone. Driving a Steinmann pin through the hard cortical bone in the midshaft will split the bone. We should pay particular attention to the point at which our skeletal fixation transfixes the bone in order to avoid splitting.

With careful attention to these details and avoidance of distraction, this method is a valuable adjunct in the treatment of the more severe types of fractures.

Rocking of the bone on the pin I have not seen in cases in which non-padded plaster casts were used and in which the joint above and below the fracture was fixed in plaster. I can see that it could occur even in a long cast in the case of padded plaster of Paris. In order to get weight-bearing on the fracture site during ambulatory treatment, we should avoid distraction. Before we put the plaster cast on, traction is released enough to get firm, hard contact of the bone ends, not impaction but pressure. The upper pin, at least, should be removed at the earliest possible moment.

I think this is an extremely valuable paper, and I hope it will be widely read and accepted, because abuse of skeletal traction may bring it into disrepute.

John A. Caldwell (Cincinnati): I heard nothing to which a person experienced in the use of skeletal traction could take any serious objection. My purpose is rather to comment on some of the skeletal traction procedures, and particularly on the excessive use of the method. Personally, I have not been able to see any particular reason for the use of a Kirschner wire. The difference in diameter does not particularly reduce liability to infection. The Steinmann pin is simpler, it is cheaper, it is put through with less formality, and it is a safer thing to use; and it does not require an expensive and complicated contrivance to keep it taut.

I cannot see any reason for driving a pin through. As Dr. Griswold has said, if you do it in cortical bone, you are almost certain to split the bone, and when it is rotated through cancellous bone, it engages firmly enough so that the limb does not slide back and forth on the pin.

I agree particularly with the abolition of any sort of dressing over the point of emergence and insertion of the pin. The frequent changing of those dressings, the application of antiseptics around them, leads to skin irritation, and our experience has been very much better since we abandoned them entirely.

The application that we put on in the first place for the purpose of coagulating the serum and sealing the pin tight is a solution of gentian violet.

Recently the market has been flooded with various types of distraction apparatus. The great value of this is that a person can buy it and use it and it is not necessary for him to bring to bear any basic understanding of the principles of handling fractures; in fact it is hardly necessary, according to some of the circulars, for him to cerebrate at all when employing it; he simply has to read the literature. The result is that these things have often been used, as Dr. Griswold has mentioned, when there was no excuse for them at all. Reducible fractures have been put up with two pins, and what I think is just as bad, multiple pins have been used in cases in which a clean, well-conducted, open operation, followed by the encasement of the limb in a cast, would have put the patient back on duty very much sooner.

This sudden terror of open operation where it is really indicated is difficult to understand. In the hands of a surgeon accustomed to doing this variety of surgery with the right facilities available, the results are favorable.

The popularity of distraction apparatus, of course, is just the result of a human trait to which Benjamin Franklin called attention. The man who pays a pound for his whistle is very apt to blow on it whenever he has any excuse at all.

It is hardly possible, I think, to overemphasize some of the dangers of the indiscriminate use of skeletal traction. In the general run of cases it is comparatively innocuous, but you simply cannot stick pins in bones and keep on doing it indefinitely—sometime or other you will shudder at the mere sight of a pin. Nevertheless, considering the simplicity of skeletal traction, the comfort of it after it is installed, and its effectiveness, it is obvious that it has an established and very useful place in surgery. To be

entirely just when comparing its dangers with those of skin traction one must not forget the pressure sores, ineffectiveness, cellulitis, and constant discomfort present to some degree in every case of adhesive plaster traction.

LAURIE H. McKIM (Montreal): I should like to ask Dr. Mathewson what he thinks of the use of the extension method by wire through the os calcis and whether he thinks the hazards in the os calcis greater than in other bones of the body.

DONALD GORDON (New York City): It seems to me that the situation as we have it at the present day is—There are certain fractures that we can take care of by simple manipulation and reduction; there are certain cases that have to be taken care of with skeletal traction; and there are certain cases where, if one has an adequate organization, it is imperative to go in, reduce the fracture and use an internal fixation.

Dr. Mathewson's paper is very timely. The facts that have been called to our attention about the high pressure salesmanship of gadgets are most important. The younger men, who have not had a long experience in the treatment of fractures before these gadgets appeared, consider that their problems are answered by these devices. We, who have come along the years, know that this is not the case.

In one of the first fractures I treated with skeletal tongs, a good many years ago, the tongs slipped and I was under the necessity of making the same sleeve protection for the tongs to prevent penetration of the point, that Dr. Mathewson has applied to the pin.

If one has instituted tong traction, which is embraced by "skeletal traction," there is a penetrating action from both sides, but there is also a great deal of graving action due to the pull forward. If this is a locked or fixed tong and is not watched, one tooth may penetrate one side and let the long tooth come out of the bone grip on the opposite side. With a sleeve on the tongs this is obviated just as Dr. Mathewson has said. It is excellent in principle and very desirable.

The truth is coming out regarding Steinmann pins. The Steinmann pin is essentially a drain to its bony position. On the other hand, as Dr. Robert Wagner of Pittsburgh mentioned in a paper he read in New York a few weeks ago, "It isn't the mechanical device one puts into a wound, it is the infection one carries in with it." A Steinmann pin can be introduced under good conditions, a small incision made, the skin separated, the pin put in and skin edges treated as one likes. If there is any chance of infection around the skin incision, I personally like to use grease to prevent crusting and retention of drainage. If any infection which has been carried in does not have adequate drainage, the pin is prevented from acting as a drain. When the times comes to take the pin out, it is properly prepared and cleansed. On its removal I take the little rubber tubing used to cover the ear wires of spectacles and follow the pin from one side with a piece of the tubing. As the pin is pulled out, I run the tubing through and then put in another piece on the opposite side. If there is any question in my mind

about infection having been carried in, or infection around the soft parts, Carrel technique is instituted. The tubes are so small that they have to be irrigated with a hypodermic needle. If that is a disadvantage due to the corrosive action of the solution on the steel needle, one can glue to the smaller tube with ambroid glue, a larger caliber tube which accepts a glass eye dropper. The wounds are dressed, as for a regular Carrel technique, with vaseline gauze. The wound is irrigated for several days with Dakin's solution until infection has been brought under control. Then the tubes are gradually withdrawn and, if there is any fear of remaining infection, the outside of the wound is treated with a 3 per cent salicylic ointment made from boric ointment without the wax, to prevent crusting. Up to the present, I have not had any difficulty from these wounds with either early or late infection.

Regarding distraction, Dr. Robert H. Kennedy of the Beekman Street Hospital, New York City, stated that when they get a distraction with Russell traction, they get delayed union. Distraction means that the muscles have lost their tone. As I see it, there is only one way for such muscles to regain their tone, and that is by the patient's activating them. The muscles of a limb can be activated if that limb is enclosed in a solid cast. There may be no visible movement of the joint, but the patient is taught to activate his muscles. It is helpful to his muscle sense to have him activate the same muscles of the other leg. There will be a certain degree of preservation of muscle tone in those muscles whether they are in a cast or in a Russell traction, tending to prevent and even overcome slight distraction.

A. WILLIAM REGGIO (Boston): There is one more thing that might also be included in the misuse of skeletal traction, and that is a mistake seen frequently, made by men treating subtrochanteric fractures of the femur. They pull the daylight into (as well as out of!) the fracture, forgetting that in this type of fracture you have to bring the distal portion of the fracture to the proximal, because of the rotation, abduction and flexion of the proximal fragment. This simply means that they do not recognize the muscle pulls in the fracture. We see only too often that they expect to obtain reduction with the leg kept straight in bed instead of in rather wide abduction and flexion. The position of the limb is of great importance in the efficiency of skeletal traction.

CARLETON MATHEWSON, JR. (closing): I should like to thank the gentlemen for their comments.

Dr. Griswold says that he has never seen the rocking motion in the upper third of the tibia. I feel very happy for him, because we have used both padded and non-padded plaster casts and have found that the non-padded plaster will not prevent this motion. I have been able to demonstrate this motion to students by simply cutting a small window over the fracture site and requesting the patient to rock up and down in bed. With motion of the body, anterior-posterior motion takes place at the site of fracture.

I agree with Dr. Griswold that when we do use two pins for traction and countertraction it is wise to remove the upper pin as soon as possible. The "proper time" is that time when the fracture site becomes sticky. We then take out the upper pin and occasionally allow weight bearing on the limb.

Dr. Caldwell spoke of the Steinmann pin versus the Kirschner wire. I think that this is like talking about catgut versus black silk and prefer not to discuss the question further except to say I like a small caliber pin because the initial damage to the bone is less.

Dr. McKim asked if I thought the hazards were greater in the os calcis than elsewhere. I can only say that if you recognize the hazards associated with skeletal traction and prevent them, you are justified in using it whenever indicated. I believe that we have not seen any more trouble with pins in the os calcis than elsewhere when we have been careful to adhere to the principles associated with the use of skeletal traction. We must prevent motion of the pin and unless we do we will get into trouble. It makes little difference how small or how large the pin, motion eventually will cause infection.

Dr. Gordon spoke of tongs. I have written of them in my paper. They are mentioned only to be condemned because even in the hands of experts tongs are exceedingly dangerous. We use the same method described by Dr. Gordon when using tongs on the skull to prevent penetration deeper than the outer table.

I quite agree with Dr. Gordon regarding the necessity of proper physiotherapy for the muscles. In all cases in which it is possible to get the patient to early weight bearing in non-padded plaster casts, we can preserve the motion of their joints even though they are unable to move in the plaster.

Dr. Reggio spoke of subtrochanteric fractures. I might add that you must not forget that the relaxed hip goes into complete external rotation. Unless you externally rotate as well as abduct the distal fragment, you will not get proper reduction.

TREATMENT OF INTERTROCHANTERIC FRACTURES OF THE FEMUR BY MODIFICATION OF RUSSELL BALANCED TRACTION*

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THE ideal contrivance for fixation of a fracture should meet the following requirements:

1. It should hold the fragments in apposition and not give discomfort.

2. It should (as a rule) immobilize the joint above and below the fracture.

3. It should relieve discomfort.

4. It should permit inspection of fingers or toes in order to determine if circulation has been impaired.

The above requirements are as old as fracture surgery itself and meet with practically uniform acceptance in principle if not in expression.

Beginning with Hodgen's suspension splint, then the adoption of the Metz or Balkan frame, the advantage of suspension in counterbalance has gradually been accepted, until now a fifth requirement may be added as a sine qua non for comfortable management of a broken bone.

5. The apparatus must move with the patient. When this condition is met, the patient can shift his position in bed to have his bed linen changed—to be put on a bedpan, to be bathed, without having the relative position of fragments changed.

In 1924 Russell of Melbourne described what was soon recognized as an extremely simple and uncomplicated form of balanced traction. Having the same weight supply both traction and counterbalance removed one troublesome feature which often mystified house officers and nurses.

The intertrochanteric fracture is one of the problems in bone surgery. Similar in age, incidence, and causative trauma with the intracapsular fracture, it is associated with the same nursing problems, such as obesity, incontinence, tendency to decubitus, and

mental states which make for noncoöperation. In many cases systemic states prevent an abduction cast, and comminution of the great trochanter may make it unfeasible to employ pins, screws
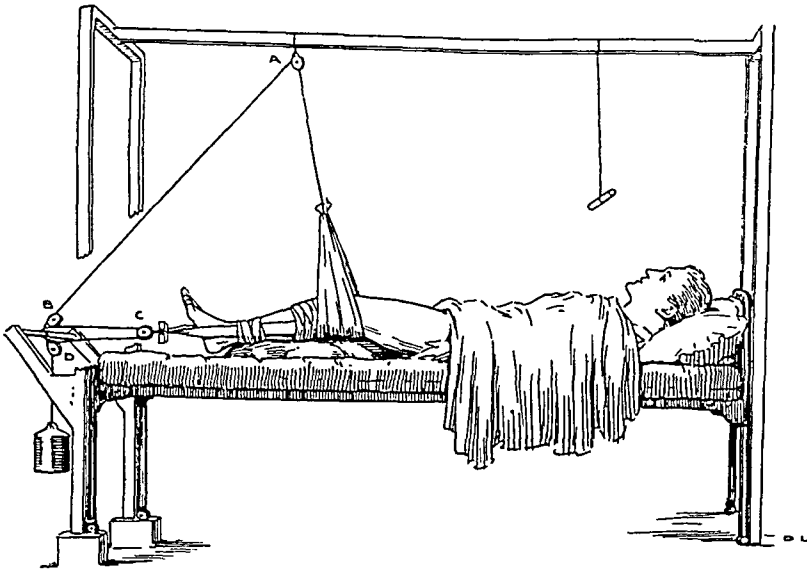
FIG. 1. Classical Russell traction.

or any direct fixation. Many of the patients have but a short time to live, but during this period of survival they must be nursed, kept clean, and cared for without torture.

It is in this group that we found the Russell traction a great boon. The patient can sit up almost straight in the Fowler position, can have his back cared for, his bed linen changed, and use a urinal or bedpan, without causing displacement of fragments.

We had used Russell traction but a short time when we recognized the many advantages it had to offer, but we also discovered two undesirable features: (1) Prolonged use of traction by means of adhesive plaster cannot be maintained in many elderly patients because of dermatitis. (2) Russell's form of balanced traction suspends the leg in a sling about the knee. Such suspension causes some pressure on the popliteal artery which, combined with elevation of the limb, imperils a circulation already impeded by sclerosed arteries. A third objection noted was that rotation of the leg could not be controlled when Russell's traction was employed.

To meet the objections enumerated above, we devised a modification of Russell's traction, which did not sacrifice any of the principle employed nor complicate its use. Practically the sole change was in substituting skeletal traction for adhesive plaster.

For a number of years we have used the Steinmann pin almost exclusively for skeletal traction. It is much cheaper and simpler, and just as safe as Kirschner wire—and is also cheaper and much
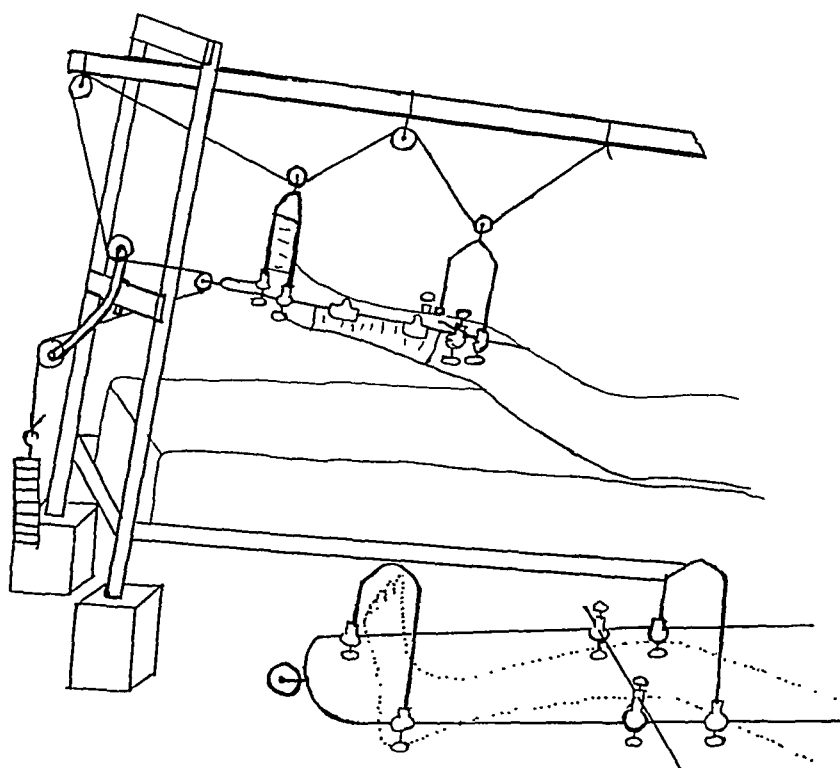
FIG. 2. Modified Russell traction showing the use of pins and lugs, with leg in the metal loop.

more safe and certain than ice tongs. Our pins are made from $5/32$ stainless steel which we buy in lengths, and have cut and sharpened in our own shop. When we use pins with a Thomas splint, we customarily clamp a special lug to either end of the pin by means of a set screw, which in turn is fastened to the bar of the Thomas splint with another set screw. Traction is then made on the end of the splint. The lugs are also useful to fix segmental fractures by means of multiple pins clamped to the side bars of a Thomas splint.

In order to use skeletal traction with the Russell principle a piece of $5/16$ inch steel is bent into U shape slightly wider than the leg, and 6 inches longer. A pulley is attached to the bottom of the U. A Steinmann pin is passed through the tibia at the tubercle and each end fastened to the side bars of the U.

Most of our Steinmann pins are inserted under local anesthesia— and the fracture is also frequently rendered painless by injecting

2 per cent procaine into the hematoma about the break. In the use of local anesthesia in fracture work we insist that no manipulation or puncture which might cause pain shall be begun until at least ten minutes have elapsed since the injection. The pin is always inserted by rotating—never driven. It is usually chucked in the ordinary mechanic's breast drill.

While our asepsis is rigid, no great formality is observed. Skin is sterilized over a small area—the operator does not scrub—and the pin is the only instrument boiled. Draping is with towels only, and the insertion of the pin is often carried out in the patient's bed. When the pin has been fixed in position, no dressing is applied where it enters or emerges from the skin, but a small area of the skin about the pin is painted with gentian violet. For some time we carefully covered the pin and the skin near it with alcohol dressings which were changed daily, but this regimen was followed by rather a high proportion of dermatitis and occasional skin infections. We then changed to an application of 2 per cent tannic alcohol with no dressing, and this was followed with but very few instances of infection. Since we have been using gentian violet, skin infection has almost disappeared. We feel that movement of the bone on the pin is a great factor in predisposing to infection.

In the 103 cases of modified Russel traction in which a pin was used, there was mild infection in twenty-one cases, and in three cases more severe infection with some degree of osteomyelitis. In all of these infection promptly subsided after removal of the pin, so that in none was there any permanent serious sequel.

When it is considered that a pin was used for an average period of fifty-one days in those patients who survived, and for twenty-one days in those who failed to survive the treatment, the employment of a Steinmann pin as a means of skeletal traction cannot be considered a hazardous procedure. In considering the hazard of skeletal traction, if one is to be just, it must be remembered that traction by adhesive plaster is almost constantly associated with minor discomfort, often sufficient to cause its abandonment, while occasional serious sequela are seen—examples are two cases of gangrene of the leg, two of cellulitis, and three of food drop from peroneal nerve pressure.

A statistical analysis follows of 103 cases treated during the five year period of the years 1933–1937 inclusive. Those cases treated in 1938 were too recently seen to justify a fair appraisal of their outcome. The cases were all from the Cincinnati General Hospital, a tax-supported charitable institution. In many of the patients who

are brought to such an institution the factors which make for poor surgical risks are present to an abnormal degree: senility, indigence, undernourishment, alcoholism, and mental impairment. Bearing these facts in mind there is abundant explanation for the mortality rate of 30.23 per cent.

AGE

	No. of Cases	Average Age
Total..	103	70.41
Males......................................	44	67.64
Females....................................	59	72.49

RACE AND SEX

	Female		Male	
	No.	Per Cent	No.	Per Cent
White.....................................	54	52.42	5	4.85
Colored...................................	40	37.86	4	3.88

SIDE OF FRACTURE

	No.	Per Cent
Right.......................................	58	56.3
Left.......................................	45	43.7

As regards the time factor from initial injury to the application of modified Russell's traction, there was a variation of from one hour to twenty-eight days.

Infection about the modified Steinmann pin inserted in the upper third of the tibia was present to a slight degree in twenty-one cases, and to a marked degree in three cases, giving a total of twenty-four, or 10.33 per cent. In the three cases showing severe infection there was definite osteomyelitis at the site of the pin, one of the three being very extensive and the others being of a slighter extent. All three healed.

The average duration of modified Russell's traction in seventy-two recovered cases was 51.19 days. The average duration of modified Russell's traction in the thirty-one patients who died was 21.34 days.

The average length of time in bed after the removal of traction in the recovered cases was 24.71 days. The average length of time in

bed after removal of traction in seven of the cases which died was 22.45 days.

The average total hospital days in the seventy-two cases which
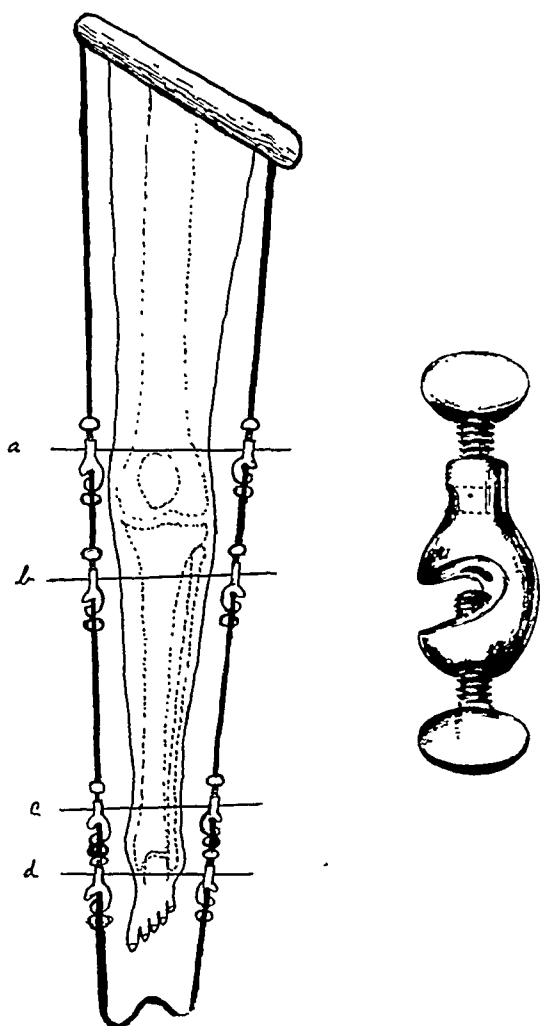
FIG. 3. Pin lug, showing various points of insertion
of Steinmann pin and fixation of pins to the side
of Thomas splint with lugs.

recovered was 87.89 days. The average total hospital days in the thirty-one cases which died was 28.16 days.

Other forms of treatment instituted after discontinuing modified Russell's traction were: (1) position of comfort—knees flexed; (2) anti-eversion boots; (3) body spicas; (4) physiotherapy.

The general mortality rate of the entire series was as follows:

Number of cases..................................... 103
Number of deaths.................................... 31
Mortality rate...................................... 30.1 per cent

Year	No. of Cases	Deaths	Per Cent Mortality
1933..	18	4	22.22
1934..	22	5	22.73
1935..	28	8	28.57
1936..	19	10	52.63
1937..	16	4	25
Average...	..	..	30.23

The causes of death in the thirty-one cases were as follows:

	Number of Cases	Percentage of Total Deaths	Percentage of Total Cases
Bronchopneumonia............................	24	77.45	23.28
Coronary occlusion...........................	3	9.67	2.91
Pyelonephritis................................	1	3.22	0.97
Head injury..................................	1	3.22	0.97
Pulmonary embolus...........................	1	3.22	0.97
Unknown.....................................	1	3.22	0.97

The results in seventy-two cases, according to x-ray evidence of anatomic position were as follows: .

	No. of Cases	Per Cent
Good...	52	72.2
Fair...	13	18.1
Poor...	7	9.7

"Good" signifies x-ray evidence of satisfactory reduction with no overriding or displacement. "Fair" signifies slight overriding or malposition. "Poor" signifies displacement or overriding.

RESULT IN SEVENTY-TWO CASES ACCORDING TO FUNCTION

	No. of Cases	Per Cent
Good...	59	82.0
Fair...	6	8.3
Poor...	2	2.8
Unknown...	5	6.4

"Good" signifies slight or no shortening with no impairment of function. "Fair" signifies moderate shortening with slight impairment of function. "Poor" signifies considerable shortening with definite impairment of function. "Unknown" signifies no follow-up.

RESULT IN SEVENTY-TWO CASES ACCORDING TO X-RAY AND CLINICAL EVIDENCE OF UNION
(CALLUS)

	No. of Cases	Per Cent
Good..	57	79.2
Fair..	12	16.6,
Poor..	3	4.2

"Good" signifies normal response of callus. "Fair" signifies delayed union. "Poor" signifies non-union.

In the seventy-two patients who recovered there was no correlation between the presence of syphilis and callus formation.

	Negative Wassermann	Positive Wassermann	No Report on Wassermann	Total
Union (good)...............	44	4	9	57
Delayed union (fair).........	8	3	1	12
Nonunion (poor)............	3	..	..	3

CONCLUSIONS

1. Certain intertrochanteric fractures, notably those which cannot be pinned or encased, can be most comfortably treated in some form of balanced traction.

2. The Russell traction is simple and effective, but unsuitable, because (1) rotation cannot be controlled; (2) the pressure of the sling on popliteal vessels with the leg elevated imperils circulation; and (3) prolonged traction with adhesive plaster is not well borne.

3. Traction by means of the Russell method modified as described is comfortable and meets the objections enumerated.

4. Skeletal traction is most simply and effectively made by means of a Steinmann pin. The pin fastened to the supporting frame by means of the lugs described makes a simple and comfortable arrangement for support and traction.

DISCUSSION

HENRY C. MARBLE (Boston): I think this is a very interesting paper. In 1915 or thereabouts, I visited Dr. Chutro in Paris. To obtain traction on the leg, he inserted a flat wire over the os calcis. This was called a Finochetti stirrup. The knee was flexed and the hip was flexed. There was no suspension or Thomas splint. The lower leg rested upon two pillows.

Shortly after, the Steinmann pin came into being. We drove this pin through the os calcis.

One day in about 1922, a kindly gentleman visited our clinic at the Massachusetts General Hospital. During his visit he drew on the back of an x-ray folder a small diagram of a new method of traction. It was a parallelogram in which the various lines of force were carefully worked out. When it was all over, I quietly inquired his name, and he said he was Dr. Russell. We tried his traction rather diligently without a great deal of success, and discontinued it. We then went back to the Thomas splint. About that time we had a patient admitted to the ward who was very feeble, had diabetes, and required large doses of insulin. She had fallen and had an intertrochanteric fracture of the femur. Despite our diligence, she died at the end of the fifth week. At autopsy it was found that she really died of inanition.

We took out the specimen of the hip. It so happened that just about that time there was a clinic in Dr. Harvey Cushing's hospital and Dr. Star of Toronto visited us. We all took the specimen in our hands and tried to bend it. It was impossible to get any motion at all at the site of fracture.

All of this led me to believe that intertrochanteric fractures of the femur heal rapidly, thoroughly, and completely, and are sufficiently united in four or five weeks. Traction is the essential in the treatment of the intertrochanteric fractures. Russell used suspension and got good results. Chutro did the same with the Finochetti stirrup. Steinmann accomplished the same thing with his pin.

Dr. Caldwell has now united the Steinmann pin and the Hodgen's splint to get the same results. That is all that is necessary. This is an excellent and very commendable addition.

I want to say one more word on the matter of internal fixation. In view of all this, why anyone should want to subject these poor, unfortunate people to pins, nails, spikes and screws when the fracture is going to heal in four to five weeks with a little well applied traction, I cannot understand. I do not think that internal fixation is indicated or necessary. Don't misunderstand me—in an intracapsular fracture, I am strongly in favor of internal fixation. In a subtrochanteric fracture there is a different problem. But this .particular problem—the intertrochanteric fracture—can be treated by simple, effective traction, and I do not think internal fixation is called for.

PRESTON A. WADE (New York): Dr. Caldwell has given us something of great value. He has limited his method to cases which are not suitable for internal fixation or other methods of treatment. I know he uses internal fixation in some intertrochanteric fractures.

In the last few years, at the New York Hospital and on one service at City Hospital, we have been using internal fixation in certain cases of intertrochanteric fractures in which there was no comminution of the greater trochanter where we feel that the nails will not cause splintering of the bone and loss of position. We have used the multiple pins, the Moore nails. We have never used the Smith-Peterson nail in these cases because of the danger of splintering the upper end of the lower fragment. We have been

careful to choose those cases in which we feel that the internal fixation will be satisfactory.

So far we feel that this method of treatment is of value, chiefly because the patients are kept in bed only a few days, are allowed up on crutches early, and do not have joint disabilities in knee and ankle.

I know there is a difference of opinion about whether these cases should be treated by so-called radical treatment. We feel that internal fixation in some of these cases is really conservative treatment, more conservative than fixation in a plaster or traction.

We have had one unfortunate experience after nailing one of these fractures in an older person. The patient was not able to use crutches very well. She slipped and fell and fractured her femur below the point of internal fixation, because the long, oblique fragment could not stand the strain of the added weight.

In cases unsuitable for nailing, we have employed Russell traction, and have used it with considerable success. We must admit its disadvantages, as Dr. Caldwell has outlined. In some patients, particularly older people whose skin cannot stand long continued traction by adhesive, infections result from skin irritation. In one case a rather severe cellulitis developed in the leg and was followed by a suppurative arthritis in the ankle, necessitating amputation.

We have had three cases of foot drop from improper application of the adhesive in Russell traction. Since we have had these complications, we have been very careful to protect the peroneal nerve.

We have encountered some irritation of the skin in the popliteal space, but have not seen any severe damage to the circulation.

Russell's original article in 1934, in which he described his method, referred to frequent criticisms in regard to eversion. He stated that the sling under the knee prevented eversion and that he had had no trouble at that time with external rotation. However, it is true that one does have difficulty with external rotation of the lower fragment, and Dr. Caldwell has given us a method which overcomes this objection.

W. L. ESTES, JR. (Bethlehem, Pennsylvania): I have been very much interested in Dr. Caldwell's paper because we have been using a modification of the Russell traction apparatus largely because of the difficulty in controlling the upper fragment in fracture of the femur by the usual *simple* Russell traction. Feeling that it is desirable in many instances of fracture of the middle and upper thirds of the shaft of the femur, we have used the Russell traction in connection with the Pierson attachment of the old Thomas splint. In that way the position of the entire lower extremity can be properly controlled in any desirable abduction or external rotation. The lower fragment can be brought in alignment with the upper, and still maintain the Russell principle type of traction. This we have found efficacious in a few intertrochanteric fractures, in fractures of the middle and upper thirds of the shaft of the femur, but we have not found Russell

traction satisfactory in fractures of the lower third of the femur, and have felt that some other method of traction should be used in this location.

KENNETH M. LEWIS (New York City): I have been much interested in both Dr. Caldwell's method and the method Dr. Estes brought out. On the other hand, one of the fundamental principles of Russell traction is putting the muscles at physiologic rest. One reason for getting away from elevating the leg in a Thomas splint with a Pierson extension and flexing the thigh too much was the fact that this throws certain thigh muscles into spasm and defeats a good deal of the traction. The question of physiologic rest of the muscles, slight flexion at the hip and slight flexion at the knee, is just as important as the mathematical calculations that go into the construction of the parallelogram of traction arrangement.

We have had ninety-eight intertrochanteric fractures on the fourth surgical division at Bellevue Hospital during the past five years. We have used Russell traction routinely and have sensed excellent results. We feel that it is extremely important in any of these methods to apply traction early—as soon as the patients are admitted, even if this is 3 or 4 o'clock in the morning. We have been able to reduce them all, and as Dr. Marble says, unions are practically 100 per cent. We have had one nonunion in the series of ninety-eight cases.

I would like to mention that we have also had the same difficulty in rotation of the fragments in fractures of the tibia. We have entirely given up the two-pin method of treating tibias whether it is the Anderson apparatus or one of the allied methods.

JOHN A. CALDWELL (closing): Regarding the lines of force with the suspension in two places with the pulley—with the leg level and the thigh slightly flexed, if the principle of parallelogram of forces is applied, it will be found that the line of traction is in the long axis of the femur. I do not think there is any change in the direction of pull in this modification over that in the original one described by Russell.

We have not employed this method for treatment of fracture of the femur, preferring the Thomas splint with the Pierson attachment, an arrangement similar to the one which Dr. Estes has mentioned, except that we also use a pin through the tubercle of the tibia to make the traction. That gives a little more support to the shaft and somewhat greater flexion of the knee and of the thigh. The suspending force is not the same one which makes the traction.

Really, this paper should hardly be called a method of treatment of intertrochanteric fractures. I think a better title would be "a method of nursing patients with an intertrochanteric fracture." The whole purpose of this is to counterbalance the patient and put him in such a comfortable position that he can be changed and taken care of without being harassed.

What Dr. Marble said about union, of course, is quite true; these fractures practically all unite, but the trouble is during the period before they have united, unless they are put up in some way that allows com-

fortable nursing, the patients are tortured quite a bit. For that reason and that reason alone, we prefer internal fixation when there is no contraindication. The patient who can have his femur nailed can be gotten out of bed in a week or ten days. That is much better than any method that keeps him bedridden for from six to twelve weeks.

The method of Dr. Estes is similar in principle to the Russell traction, except that it is a little more complicated. The suspending force is not the pulling one.

A SIMPLE EFFECTIVE METHOD FOR THE TREATMENT OF FRACTURES OF THE UPPER TWO-THIRDS OF THE HUMERUS

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MONTREAL, QUEBEC

IT is not my intention in this contribution to claim any originality in the treatment of fractures involving the upper two-thirds of the humerus. As the title indicates, I wish simply to describe a method which has proved satisfactory in our hands over a period of about twenty years. No attempt is made to present a statistical study of end results; I wish, however, to make the statement that in no case during the past twenty years have we had cause to regret the employment of the method. There has been no case in which important loss of function of either shoulder or elbow joint has occurred, and non-union has been encountered in not one case treated by this method in our clinic.

Although the title should include fractures of the anatomic neck and of the great tuberosity of the humerus, the method described has not been used in the treatment of these cases. Since fractures of the anatomic neck occur almost exclusively in elderly people, and since each individual case must, I believe, be considered to be a special problem and the treatment thereof individualized, no routine method of treatment of this fracture can, I believe, be recommended.

Fractures of the surgical neck are common injuries. Fortunately, as a rule, comparatively little deformity is present. Also, it will be agreed, I believe, that in those cases in which any considerable deformity is present it is marked. In such cases of extreme deformity associated as it is with displacement upward and inward of the upper end of the lower fragment, I believe open reduction should be undertaken after but a short attempt at reduction by traction, sometimes assisted by manipulation under an anesthetic.

It will be noted that I do not purpose taking up the matter of fractures through the lower third of the humerus. Such fractures are in my opinion frequently difficult to handle and cannot be treated in a routine manner. The method described in this con-

tribution may frequently be properly used as a convalescent apparatus but is not adapted to the early treatment of such cases.

In all other cases of fracture of the surgical neck and fracture

FIG. 1. Anterior view of patient with axillary pad fitted. Note amount of abduction, method of fixation and projection forward.

FIG. 2. Application of plaster completed. Gutters are being fashioned. Note position of pad covered with plaster.

of the middle third, whether oblique or transverse, the method which is described has been employed routinely. Fractures in the middle third of the humerus are either transverse or spiral. Fortunately, interposition of tissue is unusual, but obviously when it is encountered operative interference for removal of the tissue lying between the fractured ends must be undertaken.

A triangular shaped, firm pad is made of sheet wadding tightly compressed by means of a roller gauze bandage. This pad is sufficiently deep from top to bottom to press against the lower borders of the anterior and posterior folds of the axilla and to reach to the level of the forearm when the latter is flexed across the patient's body. It is sufficiently wide at its base to result in abduction of the arm at the shoulder to about 15 degrees—this amount of abduction is, I believe, sufficient to relieve the pull of the supraspinatus muscle and to permit contraction of the deltoid. From before backward the pad is sufficiently long so that, when lying flush with the back and the posterior border of the arm, it will reach sufficiently far

forward to project over the flexed forearm. The pad is fixed into the axilla by means of adhesive strips attached to the apex of the wedge and carried over the tip of the shoulder. (Fig. 1.)

FIG. 3. Anterior view, indicates direction of pull.

FIG. 4. Lateral view, indicates position of fulcrum and traction downward of arm. Position of incorporated coaptation splint is indicated by broken lines.

Three of four layers of sheet wadding are carried about the body under the opposite axilla outside the injured arm and over the shoulder on the injured side and so down under the pad so that the latter is kept firmly pressed into the axilla during the application of the plaster.

The upper part of the torso, the shoulder on the affected side, and the arm are encircled by 6 inch plaster bandages. A certain number of turns are carried from the tip of the shoulder down under the pad so that the under surface of the latter is covered with plaster. A coaptation splint made of one 4 or 6 inch plaster is applied from the root of the neck over the tip of the shoulder and down to the lateral condyle. This is incorporated in the circular bandage which, as indicated above, is applied outside the injured arm and around the body. Such a dressing is not heavy, and approximately five or six 6 inch bandages suffice for the purpose.

Just before the plaster sets it is moulded to the anterior and posterior surfaces of the arm with the heel of the hand in such a

way that two gutters are produced which prevent movement forward or backward when the patient bends or lies down. (Fig. 2.)

A wrist sling is applied so that, the elbow being free and the

FIG. 5. Felt pad in position. This increases traction power and protects forearm.

arm unsupported, a certain amount of traction is exerted downward by the weight of the arm distal to the point of fracture and the greater part of the forearm. Traction is then increased to any desired extent by straining the upper border of the forearm against the slightly convex lower border of the axillary pad. (Figs. 3 and 4.) If such straining is carried out before the plaster has entirely set, a shallow groove is produced into which the forearm fits. After the plaster is dry a thin felt pad is placed between the moulded plaster of Paris on the lower border of the axillary pad and so protects the forearm. (Fig. 5.) By pulling upward on the wrist by means of the sling any desired amount of traction may be obtained. It should be pointed out in this connection, however, that there is more danger of using excessive traction in this way than of using too little.

Since on the one hand it is not usually possible, particularly in stout persons, to apply plaster to the body when the patient is under an anesthetic, and since it is but infrequently necessary to employ an anesthetic to bring about reduction by this method of traction, it has been my habit to prepare the patient by means of a

mild sedative and, should more difficulty than usual be anticipated in bringing about reduction, to employ local anesthesia by the hematoma infiltration method.

The patient is advised that for the first couple of nights he will probably be more comfortable if he does not attempt to sleep lying down but rather stays in a large comfortable chair or propped up in bed; he is informed that it is inadvisable to place a pillow under the affected arm. Sedatives, as indicated, are prescribed. I have made it a practice to inform the patient or his guardian when first seen that, after a few days, considerable swelling, with probably discoloration and soreness about the elbow, is likely to be exhibited and should be expected. If this is not done the patient is likely to be alarmed when these phenomena, which are due to the seepage of blood from the site of fracture downward, has taken place.

Approximately seven to ten days after injury it is advisable to permit the patient to take the forearm out of the sling and, supporting the hand on the injured side by means of the uninjured member, commence active movement of the elbow joint. It is recommended to the patient that eight flexion movements be carried out in this way three times a day and that care be exercised that pain is not produced. From the very moment of injury it is wise, I believe, to urge the patient to exercise his fingers and wrist and to carry out the rotary movements of the forearm. Such advice avoids stiffness of these joints and results in a more adequate blood supply to the limb as a whole.

Second only to the quadriceps extensor of the thigh the deltoid muscle atrophies more quickly, I believe, than the other muscles of the body when put on the stretch and unused. Delay in return of function of the shoulder joint is most surely avoided by prevention in so far as possible of deltoid atrophy. In order that such atrophy may be minimized exercises are commenced about ten days from the date of injury. Although actual movement of the shoulder joint is prohibited by the retaining apparatus, both the supraspinatus and the deltoid can be induced to contract against the fixed resistance of the plaster, so that the strength and mass of the muscles are maintained.

Fractures of the surgical neck of the humerus heal comparatively quickly; depending upon the age of the patient the fixation apparatus may be removed in from two to five weeks from the date of injury. Following removal of the plaster the patient is supplied with a cravat sling with instructions to employ the limb actively so far as it is possible without pain and, at the same time, to continue

purposeful exercises of the muscles of both the arm and the shoulder girdle. As a rule, two to four months are required for substantially complete return of function to be obtained although at the end of six weeks from the date of injury it is usual to have reasonably good function of the arm exhibited.

Since it would seem that the same force is responsible for fracture of the surgical neck and of injury to the supraspinatus tendon, that is to say, to the floor of the bursa, it is not surprising that in some cases a prolonged period of disability due to the bursal injury is noted. When musculospiral (radial) nerve injury complicates the fracture, the lower half of the forearm and hand are fixed in the cock-up position and the apparatus otherwise applied as indicated.

Fractures through the middle third of the humerus require a longer period for union to occur than those through the surgical neck. It is possible with the apparatus in position to obtain adequate x-rays, to follow the course of calcium deposition and, also, to note clinical union without removal of the plaster. In the case of fractures through the middle of the bone, particularly of the transverse type, I have been accustomed to leave the apparatus undisturbed for a period of about eight weeks at which time it is removed and a careful check up of the situation carried out. Since in our experience the majority of musculospiral (radial) nerve lesions recover without operative interference, it has been our custom to postpone exploration of the nerve until union of the bone has occurred.

Although it is true that the typical deformity of the upper fragment in fractures of the surgical neck is one of more or less abduction and external rotation, it seems evident that, presumably due to the correcting force of the long head of the biceps when this powerful structure is made tense, moderate traction with the arm in internal rotation downward can be counted upon to bring about almost perfect reduction of fragments except in those cases in which an interposition of soft tissues has occurred. This fact has already been, I believe, clearly demonstrated by Griswold and others.

Since 1918 a number of authors have recommended from time to time the employment of the abducted position in the treatment of fractures of the surgical neck or middle and upper third of the shaft. Some have recommended the position of internal rotation, that is the so-called "airplane" position; others in external rotation, that is the so-called "traffic cop" position. For purposes of abduction, splints or plaster-of-Paris have been used as an ambulant method and traction abduction in a Thomas or similar splint, using either skin or skeletal traction, have been employed. Despite the

fact that these recommendations have been made, and despite the fact that certain of the junior members of my staff have employed such methods with, it is true in the main, satisfactory results, I have never been convinced that abduction resulted in either better anatomic position, shorter period of active treatment, or earlier or better return of function. Since, on the other hand, the abduction method is inconvenient if the patient is ambulant, and expensive if hospitalization in bed is employed, its only justification would be proof of better immediate and ultimate results.

With the exceptions noted in the introductory paragraphs the method which has been described fulfills, I believe, the qualifying adjectives employed in the title. It is simple in that it is easy to apply and it is inexpensive. It is effective in that it is comfortable for the patient and, by means of its employment, our results have been uniformly satisfactory. Since the patient is ambulant he may carry on his affairs if such activities do not require the use of both upper extremities. In any event, since hospitalization is not required for more than a few days at most, it is economical from the patient's point of view and, also, from the point of view of hospital beds.

<div align="center">DISCUSSION</div>

GROVER C. PENBERTHY (Detroit): Dr. Gurd has called our attention to the need of individual treatment for each type of fracture. He has emphasized the advantages of the plaster dressing as he has described it for fractures of the upper two-thirds of the humerus.

I have had no experience with this type of fixation or reduction, but Dr. Gurd, in his twenty years of experience in using this type of traction and reduction and fixation, has had most satisfactory end results.

The disadvantages of the airplane splint or the arm abducted with plaster fixation has been emphasized. These are well known to all of us. This method of treatment allows the patient to be ambulatory and is economical as well as effective.

The economic aspect of fractures affects us quite materially in Detroit, especially at the City Receiving Hospital. We have had on the surgical service at the Receiving Hospital some forty patients treated by the hanging plaster dressing. In addition to the surgical service, the orthopedic service also treats these fractures with the hanging plaster and within the last two years Dr. LaFerté has reported a large series of cases in which the hanging cast was used.

At the Receiving Hospital approximately forty cases have been treated by this method. In no case was there nonunion, and in only one case was there delayed union.

I can, however, see a very material advantage to this dressing described by Dr. Gurd; the moulding of the plaster on the lateral aspect of the arm

fixes the humerus and it should therefore make a more comfortable dressing than the hanging plaster. Both methods are effective, and in both the patient is ambulatory.

We are indebted to Dr. Gurd for bringing this particular type of treatment to our attention.

D. C. PATTERSON (Bridgeport, Connecticut): I want to congratulate Dr. Gurd on the simplicity of his treatment in these cases. The less complicated the restraining apparatus the more generally satisfactory will be the results. Some of the splints and apparatus advocated for fracture treatment today are pretty complicated. An editorial in the British Medical Journal, referring to Hugh Owen Thomas, pointed out, "The appliances he invented and finally perfected have not yet been improved upon— modern so-called improvements have been retrograde."

I should like to demonstrate the use of the angle plate, advocated by Dr. George Hawley in cases of fractured humerus. [Films were shown.] The first part of the picture shows the results obtained in operations on dogs, carried out at Yale Medical School under the direction of Dr. Bassum. After the plate was inserted only a simple dressing was applied and the dogs were allowed up and about without any restraining dressings. In from two to five days they were running about with the operated leg apparently as firm as before the fracture. It is also apparent from the pictures that these dogs were suffering no pain.

The case of fractured humerus was in a young man of 31, one of our hurricane victims. He had a slightly comminuted, transverse fracture in the middle of the shaft of the right humerus. X-ray showed the distal fragment displaced completely anteriorly and slightly inward, with overriding of about 1 cm. Attempts to keep the fragments in good position with different splints and plaster were unsuccessful, and seven days after the injury an open reduction with application of the angle plate was performed. After exposure, the bone ends were approximated and a marking scratch made to indicate the location of the slot. Then the ends were elevated from the wound in turn and the slot cut with the ordinary electric saw.

It seemed to me that it was easier to apply this type of plate than the ordinary flat one, for when it was placed in the groove there was no tendency for it to move, and it was an easy matter to insert the screws. These screws, like the angle plate, were made of vitallium. They were long enough to penetrate the cortex on the far side of the bone.

After closure of the wound, simple coaptation splints were applied to the upper arm, and the patient for the first few days rested his arm on a pillow. He started movement of the fingers and hand almost immediately. He had a very comfortable convalescence, with practically no pain. He was out of bed on the seventh day with his arm in a sling. At this time motion was started at the elbow and shoulder joints, and was carried out daily in a gradually increasing degree.

The patient was discharged from the hospital on the eleventh postoperative day and complete active motion was present at the end of a

month. He returned to work in five weeks, and at the end of six weeks he had free motion of the entire injured extremity and was able to hold a very large book on his outstretched arm. The after-care of this patient was carried out under the direction of Dr. Hawley, and he will tell you of the things that he considers important in the convalescent treatment.

The advantages of the angle plate appear obvious. There is great strength of repair; shortened period of treatment and disability; early motion and function of the extremity; and comfort to the patient.

The early appearance of callus, and its evident strength were due to the abundant circulation provided by early motion.

GEORGE HAWLEY (Westport, Connecticut): It is a great pleasure to be here, to listen to these papers, and to meet so many old friends.

Dr. Patterson wished me to say a few words about the bone plate which he used in the case he has described.

Nineteen large dogs were used in the experiments at Yale. Twenty-three femurs were plated. Seven were failures, one was infected, and in six, the bone cracked and pieces fell out a few days after operation. A dog's femur is, however, not a good test object, because the cortex is thin and brittle.

Thirteen humans have been treated with the angle plate and fifteen bones plated. Four were fractures of the humerus, two fractures of both bones of the forearm, four of the tibia, three of the femur. It has been found that screws get the best hold when tight in the distal cortex and loose in the proximal cortex. To accomplish this, a small drill is used through both cortices and the hole in the proximal cortex enlarged with a carpenter's awl. The hold of the plate depends on the screws. Protruding screw ends cause no discomfort or sensation.

In the human cases up to the present, no screws or plates have become loose. There has been new bone deposit as shown in radiographs two weeks after operation. Callus formation has been steadily progressive, as evidenced in the x-rays. The plated bones have been strong and continued strong from operation until union was complete. There has been absence of pain and tenderness at the site of fracture after wound healing.

I would like to quote two short paragraphs from a paper soon to be published: "In conversation with a writer during a visit to his hospital in Compiègne during the war, Carrel expressed the belief that when the process of fracture repair is better understood, fractures would unite in less time than now. The thought was suggested that it might not be so much the need of methods or agents to stimulate fracture repair as removing inhibiting factors which regard repair and release forces now held in check.

"The principle of the mobilization calls for effective and continued immobilization of the broken bone ends. The more perfect the immobilization, the closer we come to fulfilling the principle of immobilization. There may be more significance than we imagine in the difference between perfect and imperfect immobilization. It is possible that the more absolute the immobilization, the more active and positive the process of fracture repair."

Notable have been the following facts: Absence of erosion and atrophy of bone around screws; uniform and rapid recalcification of the fracture; strength in the plated bone as tested clinically throughout the period of repair; patients' confidence of strength in the plated bone; difficulty in determining when bony union is complete and new bone strong and plate unnecessary.

No claim is made for anything new. Personally, I dislike the word "new" and try to avoid it.

KELLOGG SPEED (Chicago): I wish to say a word to establish the date of this procedure. This plate was first described by Dr. Souttar in 1911 or 1912. I used it in 1914, 1915, and 1916 in at least ten cases, some involving the femur, some the humerus, and some the tibia. It is very easy to insert and the cutting of the slot is a very simple procedure with a circular saw.

I agree with all that has been said about its additional mechanical aid in the stability of the fracture, and with what Dr. Hawley emphasized about the difference between near immobilization and complete immobilization being the factor which leads to the success acquired with this plate. I had no failures with it; I had no infections with it. I merely make this statement to set down that the plate was first described and used by Dr. Souttar.

R. ARNOLD GRISWOLD (Louisville): I am very much interested in Dr. Gurd's description of his method. We have used since 1933 a similar method which I learned from John Caldwell—the hanging cast—with excellent success. Our series of fractures of the humerus of all types treated with this kind of cast numbers from 250 to 300. We have had one nonunion in that list.

I have found a misapprehension in speaking to various people about the traction cast, especially in regard to the surgical neck fractures. The traction cast will reduce most fractures of the shaft; it will not reduce those above the deltoid insertion. If the surgical neck fracture is properly reduced by traction and manipulation, the hanging cast will hold it in position if the long head of the biceps is intact.

The most effective maneuver that we have found for the reduction of the surgical neck fracture in connection with the hanging cast is a method described by Dr. F. J. Cotton and Dr. Gordon Morrison in the *New England Journal of Medicine* in November, 1934. It has the same advantages that Dr. Gurd's method has, as they are very similar. I am glad to see the trend away from wide abduction in fractures of the upper third of the humerus.

EDGAR L. GILCREEST (San Francisco): I was very much interested in Dr. Gurd's paper. In numerous papers on fractures I have tried to emphasize that we should return to the more simple methods instead of the various complicated ones requiring more and more gadgets. In fact, I believe in years to come this period will be known as the era of gadgets in the treatment of fractures.

When I was with von Hocker in Gratz, Austria in 1913, they were using in his clinic a method that Dr. Gurd speaks of this morning, with the exception that a very ingenious piece of strong cardboard was folded under

the arm and up in the axilla. It was very light but kept the humerus, as Dr. Gurd has said, about 20 degrees from the chest. It was folded in such a manner that the forearm could rest on it. It is important now to have Dr. Gurd call this simple and effective method in the treatment of fractures of the upper two-thirds of the humerus again to our attention.

I hope that this Association for the Surgery of Trauma will in the future more and more emphasize to the surgical profession of America these fundamental and basic principles in the treatment of all kinds of trauma and lead us away from the complicated methods with which our literature is becoming filled.

FRASER B. GURD (closing): I would like to say that I perhaps did not sufficiently indicate how apologetic I felt at bringing before such a body a matter which seemed to be so simple, even moronic in character. However, I feel, as Dr. Gilcreest evidently does, that the danger has been on the part of many of us to make the treatment of fractures appear to be so complicated that the men who actually must treat the fractures throughout the country have been at a very serious disadvantage. I feel justified, therefore, in putting on the program a method which might be described by the adjectives "simple and effective."

THE PROBLEM OF THE UNUNITED FRACTURE

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UNUNITED fractures may be divided into two groups: (1) those in which the bones are merely delayed in uniting, evidence of attempts at consolidation being apparent; (2) those in which the fragments are in a fixed state of nonunion and no evidence of an attempt at consolidation is to be seen. The element of time cannot be used to place these fractures in one group or the other, for some fractures are clearly in a state of nonunion within the short period of three months after the injury, whereas others show evidence of efforts at repair a year or more after sustaining of the fracture. Failure to recognize this division has led to confusion in the evaluation of treatment, for measures that may suffice in delayed union will be inadequate and lead to many failures in nonunion. Experience has taught that any ununited fracture is a foe worthy of the surgeon's best efforts; he is dealing, in a delayed union, with a subnormal physiologic condition at the site of fracture and in nonunion with a state of physiologic inertia.

ETIOLOGY

It is not within the scope of this discussion to discuss adequately the etiology of these conditions but several points should be mentioned.

Sex is of no importance but age apparently is of importance. Why is the extremely resistant ununited fracture of the tibiae encountered among the newborn and infants? This fact cannot be readily accounted for, but probably some congenital anomaly of the blood vessels is accountable. It is possible that in some adults the blood supply to certain regions is congenitally poor and, on a fracture being sustained at these sites, union fails to develop. Except for fractures of the neck of the femur, ununited fractures are no more common among elderly than among younger patients.

Patients with ununited fractures are almost invariably in excellent general condition, and no clue as to cause can be elicited on the most painstaking general examination. The cause, therefore, must be local. A history of severe, traumatizing original injury often is given

by the patient with an ununited fracture; in such a case the insult to tissues may be so great that repair is slow to develop and, when it does develop, is unable to go on to completion. Interposition of muscle between the bone ends, the muscle later becoming a fibrous mass; inadequate reduction; faulty fixation; too early mobility and, in the lower extremity, too early weight bearing, are all prominent etiologic factors. In some few cases no cause can be ascertained; the bone simply will not unite although all conditions seem favorable. Bones differ in their shape and size, also as to the functional demands made on them and the manner in which stresses of force are transmitted through the sites of fracture. All of these have more or less bearing on the reason why a given fracture fails to unite. To my knowledge, no dependable figures ever have been presented to show the percentage of fractures that will fail to unite within the normal period of healing.

GENERAL CONSIDERATION OF TREATMENT FOR DELAYED UNION AND NONUNION

Regulation of diet and administration of glandular products or drugs are of no real help. It must be recognized that physiologic inertia exists at the site of fracture and that this must be done away with and the process of regeneration of bone initiated. In the treatment of ununited fractures, as in other conditions wherein surgical treatment is difficult, a great variety of opinions as to method are to be found. Because, for example, union in a case of ununited fracture of the tibia may result on weight bearing in a walking caliper, it may be argued that in all cases such treatment should be employed. Because delayed union of the fragments of a humerus occasionally is overcome after congestive therapy and massage, it is argued that such treatment should be used. Because, in another case, union takes place after injection of venous blood into the site of fracture, that type of treatment is advocated, and one could go on citing many more methods. Advocates of such lines of treatment never have reported any sizable series of cases and, on inspection, there is little to support the majority of the claims. The same criticism applies to drilling of the ends of the bones. All these measures have some merit and may turn the tide and stimulate formation of bone in the presence of delayed union, but, in my experience, they are absolutely futile against nonunion.

It may be asked how delayed union can be distinguished from nonunion. In the typical case of nonunion, pain and tenderness are absent and the roentgenogram indicates that the bone ends are

clear-cut and rounded. They lack the irregular, somewhat fuzzy, excessive formation of callus so often seen in cases of delayed union. Often a year or more has elapsed since the injury. Such fractures require operation. Any open operation which consists of freely dissecting away the fibrous tissue from about and between the bone ends, freshening and fitting them together, and providing internal fixation by metal devices and external fixation with casts or splints, will bring about union of many ununited fractures. Numerous reports have established without doubt that the autogenous bone graft gives the best results. Therefore, my discussion as to treatment will be confined to the use of the bone graft; I shall mention briefly the general technique and the more specific technique involved in treating several fractures of the more intractable types.

SOME ESSENTIALS FOR BONE GRAFTING PROCEDURES

Repetition is necessary in order to keep constantly before surgeons certain cardinal principles as to selection of cases, technique and after-care. They can be enumerated as follows:

1. No ununited fracture should be subjected to direct operative attack if there are any draining sinuses or if there is any latent infection in the vicinity of the fracture.

2. Fibrous tissue should be removed from between and about the bone ends; the medullary cavities should be opened, and the ends of the bones fitted, so that they can be held in close apposition by the aid of a bone graft.

3. The graft must be long and large enough to insure large surface contact of graft with both fragments. The bone graft serves two functions, a physiologic function in stimulating formation of bone and a mechanical function in providing internal fixation.

4. The graft must be held securely to the fragments by aid of beef bone screws, autogenous bone pegs, metal screws or metal bands. Multiple chips of bone should be packed along the line of fracture and as much as possible along the line of contact of the bone graft with the fragments. The more bone that is brought to the site of fracture the better.

5. External fixation, preferably by casts, must be provided for sufficient time to protect the graft during the period of "weakness," which develops about the fourth to the sixth week. This protection is particularly necessary in cases of fracture of the humerus or femur. Three months is the earliest that external fixation can be abandoned and some sort of protection is often essential for six months from the time of operation.

6. It must be constantly kept in mind that repair of bone is always slower to be induced after it once has failed. The second attempt at repair is certain to be slower in starting, developing and finishing than nature's first attempt. Because of that fact careful supervision of resumption of function, as to time, manner and amount, is the definite responsibility of the surgeon.

RELATIVE INCIDENCE OF UNUNITED FRACTURES OF VARIOUS BONES

In a review of the records of 583 patients with ununited fractures treated by bone grafting operation from 1912 to 1936 inclusive in The Mayo Clinic, the relative incidence as to the bones involved was as follows: tibia, 233 cases; humerus and femur, 100 each; radius alone, seventy; radius and ulna combined, forty-four; ulna alone, thirty; clavicle, five; and metatarsal, one.[5]

RELATIVE RESULTS IN TREATMENT OF FRACTURE OF DIFFERENT BONES

In 530 cases the end results were definitely determined. The results following bone graft of the tibia were the best in that in 94 per cent of cases the fragments united. Success was obtained in treatment of fractures of other bones as follows: the radius alone in 93 per cent of cases; the ulna alone in 89 per cent; the humerus in 86 per cent; the radius and ulna in 85 per cent and the femur in 78 per cent. Difficulties encountered in dealing with individual bones differ with the location of the fracture, be it in the upper, middle or lower third.

TROUBLESOME SITES OF FRACTURE OF VARIOUS BONES

The anatomic division of the various long bones into upper, middle and lower thirds is recognized, and it is interesting to study the end results on that basis.

Concerning the shaft of the femur, the results were equally good whether the fracture was in the upper or middle third, but if it was in the lower third the frequency of failure was higher. The percentage of good results relative to the tibia was lowest when the fracture was of the lower third. The bone in that region is smaller than it is elsewhere and surface contact of the bone consequently is less. Also, the bone of the lower third is more pipelike, has less blood supply and the surrounding structures are not so vascular as is true of the upper and middle thirds. Again, fractures of the lower third of the humerus gave more trouble than those of the middle and upper thirds and the

FIG. 1. Nonunion of neck of femur eight months' duration.

FIG. 2. Same case as that represented in Figure 1.
Good alignment following ten days' traction.

results were relatively poorer than when the upper or middle third was concerned. The lower fragment is often short and the difficulty in preparing it for reception of the bone graft is great, thus necessitating
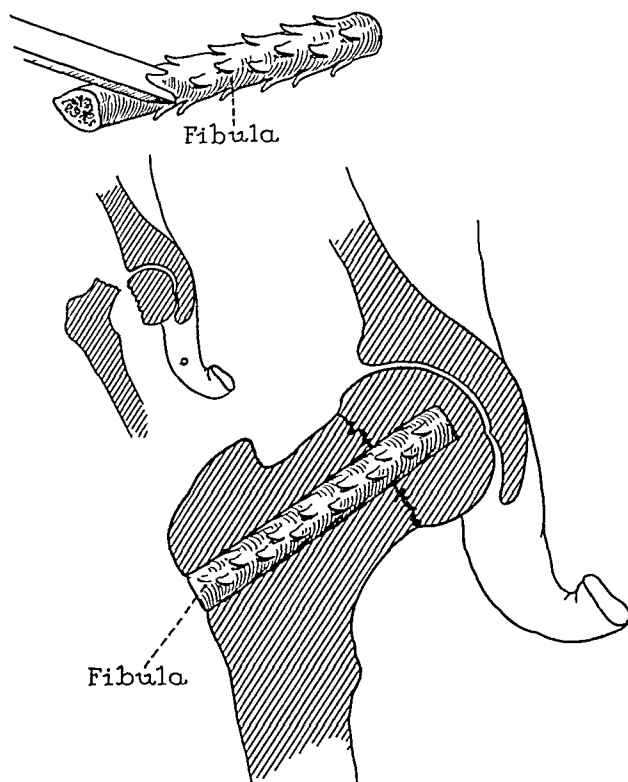
FIG. 3. Fibular bone graft cleansed of muscle and periosteum and roughened to permit more ready vascularization.

more dissection, manipulation and trauma. This is especially hazardous if operation has been performed previously with consequent formation of scar and, worse, if there has been infection. In cases in which the radius alone was involved, the percentage of failures was higher if the fracture was in the lower third than if it was in the middle or upper third, and the same was true when the ulna alone was involved.

When the operation was performed because of fractures of both the radius and ulna—in other words, because of a double fracture of the forearm—repair of fracture of the middle third gave the poorest results. In that situation, particularly in the presence of delayed union, there is often an attempt at synosteosis between the radius and ulna, necessitating extensive and time consuming dissection to remove the excessive callus. In some cases I am convinced it is better to perform the operation in two stages; first, to operate on one bone,

and after that has united to operate on the other bone. Such a procedure in two stages is objectionable from the patient's viewpoint but may be to his best interests.

FIG. 4. Same case as that represented in Figures 1 and 2
Union fourteen months after operation

TECHNIQUE IN CERTAIN TYPES OF CASES

It may be stated that, other things being equal, the larger the graft and the greater the amount of surface contact of the graft with the fragments, the better is the chance of success. This has led me to emphasize what, at the clinic, we have called the "massive"graft,[2] [3,4] which is of the same type that Campbell has aptly called the "onlay" graft.[1] If this type of graft were always referred to as the "onlay massive" graft, it would serve to emphasize the point that but little bone should be removed from the cortex and that the graft should be large.

Another point I wish to emphasize is that the packing of bone chips, and especially chips of spongy bone, about the site of fracture is of great importance. By using the massive graft, fixation is pro-

vided and the chips bring extra bone to the region where it is most needed.

Fractures of the Neck of the Femur. "The head of the femur must
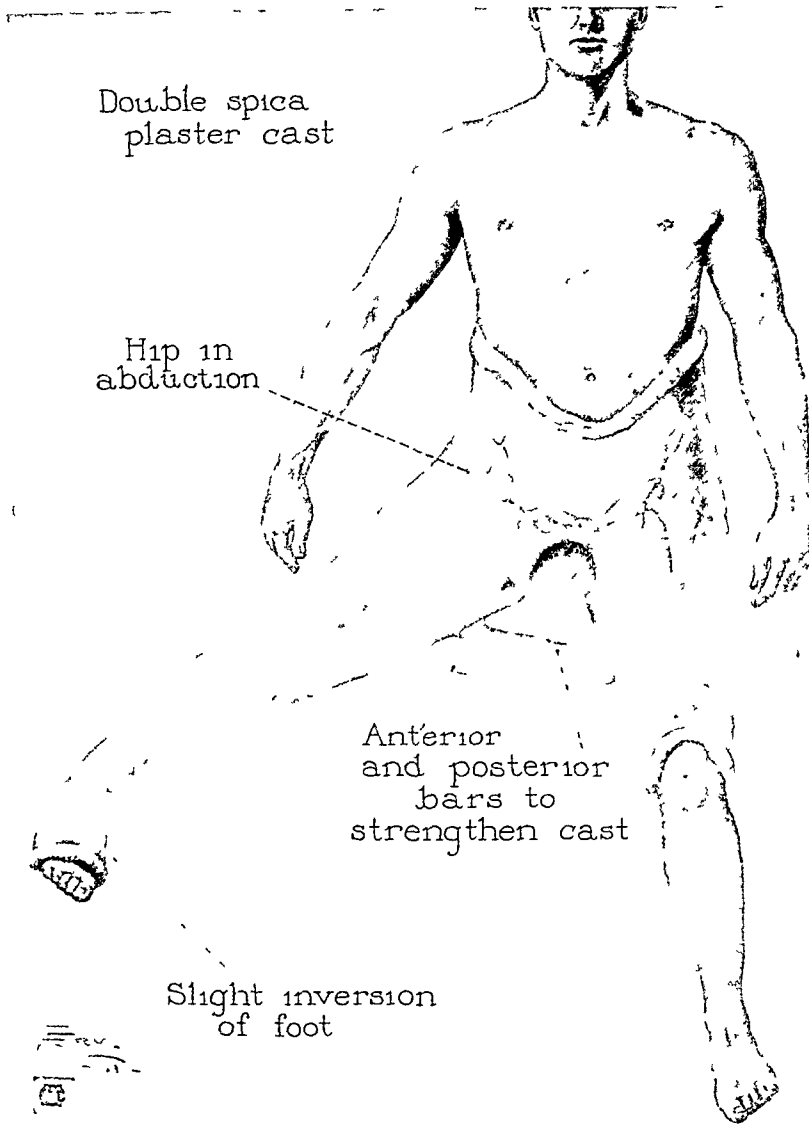
FIG. 5. *Double spica cast.*

be viable; there must be sufficient femoral neck left to work with; the fibrous tissue of the union must be dissected out, and the bone ends must be freshened and accurately fitted if the maximal results are to be obtained."[5] This is an excerpt from an article I published more than a year ago. With the exception of the remark about the

viability of the head and the amount of neck left I am not certain that statement is correct.

We have had some excellent results from "blind pegging" of the

Fig. 6. Fibular graft was inserted over guide wire without opening joint. Reinforcement by lag screw.

fibula in cases in which fibrous union was present. In these cases by skin or skeletal traction it was possible to pull the lower fragment down and restore normal anatomic relationships (Figs. 1 and 2) with good alignment of the head and neck. A guide wire was inserted under roentgenologic supervision and verification; a drill of proper size was fitted over this wire and a hole was made through the trochanter and neck and well into the head. Then a fibular segment of proper length was inserted over the wire. The fibular graft should be thoroughly cleansed of all muscle, fascia and periosteum and it should be roughened with the aid of a chisel to insure ready penetra-

tion of blood vessels from the surrounding bone. (Fig. 3.) It is possible
that the piece of the fibula acts as a physiologic stimulus to formation
of bone and that the fibrous union develops gradually into bony

FIG. 7. Nonunion of humerus with typical angulation at site of fracture.
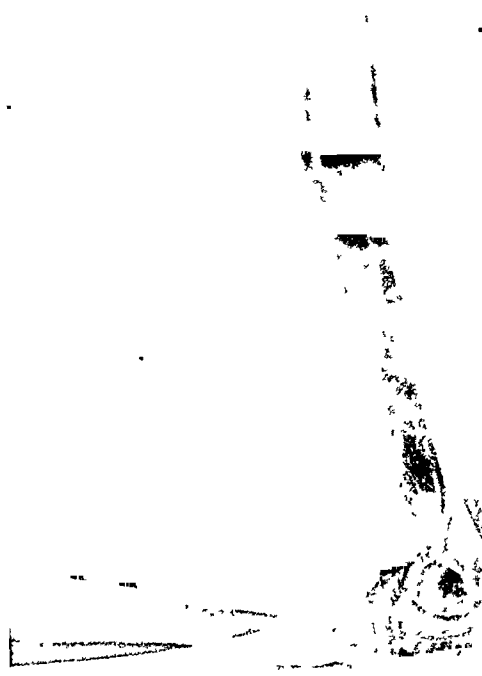
FIG. 8. Same case as that represented in Figure 7. Deformity corrected and fragments held with bone graft.

union. (Fig. 4.) In those cases wherein there is no fibrous union and
the joint fluid plays about the surfaces of the fracture, this method
probably will not be successful, for it is known that bone pegs
inserted across joint surfaces tend to fracture at the joint line in cases
in which they were used in the hope of inducing arthrodesis.

Postoperative fixation in a double spica cast is essential. Motion
should not be permitted (Fig. 5) in less than three months and weight
bearing should not be allowed until at least six months have elapsed.

In two cases, in addition to the bone graft, I have used a lag screw
to help maintain fixation. In these cases (Fig. 6) results were happy
and I was able to permit motion much earlier than in other cases. It
must be remembered, however, that the head of the femur is small
and poorly supplied with blood and that it will not remain viable
when subjected to ruthless drilling.

Nonunion of the Lower End of the Humerus.   In cases wherein the
lower fragment is short, it is usually difficult, without undue trauma,

to expose enough of the fragment to allow application of the massive bone graft. Exposure is best carried out by an anterior external incision, keeping well anterior and medial to the musculospiral nerve. Too vigorous traction may produce paralysis of the musculospiral. The median nerve lies well to the medial side and, if exposed, should be gently held out of the way.

Once exposure is adequate, enough of the cortical bone on the anterior surface of the lower fragment should be chiseled away to produce fairly free bleeding. After the bone ends of each fragment have been sawed off squarely, so as to fit accurately, and the medullary cavities have been opened, the cortex on the anterior surface of the upper fragment should be prepared in the same manner as that of the lower fragment was prepared, care being taken that the prepared surfaces are in the same plane.

The next problem is fastening of the graft to the lower fragment. This can be done by inserting beef bone screws through the graft and opposite cortex. Vitallium screws can be used. If the cortices of the fragments are too osteoporotic to allow screws to be employed, use of Parham bands may be required. It is often necessary to tilt the lower fragment well out of the wound and then put in the graft. Next, the graft is brought into contact with the upper fragment, care being used that the bone ends are pressed firmly together, end to end, before the graft is fastened to the denuded area on the upper fragment. Then the bone chips from the denuded cortices, the clippings from the medullary surface of the graft and the scrapings of spongy bone obtained from the upper end of the tibia, just below the epiphyseal region should be carefully packed about the site of fracture. (Figs. 7 and 8.)

The arm should be included in a spica cast and not disturbed even for dressings for about eight weeks unless there is evidence of drainage from the wound.

SUMMARY

Ununited fractures can be divided into: (1) those in which there is still some attempt at repair of bone—delayed union; and (2) those which are in a fixed state of physiologic inertia—nonunion. The etiologic factors are local and not systemic. In cases of delayed union conservative measures may succeed in stimulating metabolism of bone at the site of fracture, but not in cases of nonunion. Operative measures, preferably the use of an autogenous bone graft, are necessary in the latter cases. Operation should not be undertaken in the face of draining sinuses or of evidence of inflammation. The bone

graft should be as large as possible and must be held firmly clamped to the fragments. Multiple chips of bone packed about the site of fracture are of great aid. Fractures of the neck of the femur can be dealt with successfully without arthrotomy in cases in which fibrous union has occurred. The method consists in overcoming the shortening with traction, securing anatomic alignment, and inserting a large piece of autogenous bone, such as a fibular segment of such length that it extends from the trochanter well into the head. This procedure could be called "blind" insertion. It probably is not suitable in cases in which no fibrous union is present; then open operation is preferable. Fractures of the lower end of the humerus are the most difficult of all humeral fractures to deal with and are best treated by the "onlay massive" graft.

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EXPERIMENTAL FAT EMBOLISM*

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REGARDING the importance of fat embolism as a clinical entity there exists much confusion. Both clinical and experimental evidences indicate that fat emboli occur frequently, particularly in association with trauma to bone. There is good evidence that, in adults, some fat is embolized with most if not every fracture and with most surgical insults to bone. But the frequency with which these emboli cause morbidity and mortality is the subject of controversy. For although many fatalities have been reported in which the presence of fat in the pulmonary capillaries has been the only demonstrable cause of death, it has been shown repeatedly that animals will tolerate without appreciable effect much larger quantities of fat injected intravenously than could conceivably enter the blood stream in clinical cases.

Buerger,[1] Pinner[2] and Flournoy[3] have reported cases of fat embolism in which ruptured and crushed subcutaneous fat was the sole demonstrable injury. Virchow[4] and Warthin[5] have reported fat embolism following childbirth and have explained the origin of the intravascular fat as that liberated from the pelvic fatty tissue by pressure from the fetal head while passing through the birth canal.

Numerous authors have stressed the fact that fat emboli are found not infrequently at post-mortem examination of patients in whom the cause of death was unrelated to trauma. Reports indicate an incidence in this group of 10 to 15 per cent. Such nontraumatic causes include chemical poisons such as phosphorus, potassium chlorate, corrosive sublimate, phenol, carbon tetrachloride, arsenic, and its derivatives used in the treatment of syphilis, alcohol and potassium cyanide, as reported by Grondahl,[6] Burns and Bromberg,[7] Puppe,[8] MacMahon and Weiss[9] and Winogradow.[10] Other nontraumatic sources include intravenous injections of medications containing oil and a wide variety of diseases such as diabetes, tuberculosis, post-influenzal pneumonia and pancreatitis. Carrara[11] found fat embolism present in 22 per cent of cases of cardiorenal disease. Thus one of the difficulties in evaluating the clinical importance of the relation of trauma to fat embolism becomes apparent.

Injured bone is the principal source of massive fat embolism because in it are provided the three factors necessary to embolization of fat: (1) the presence of free fluid fat; (2) accumulation of this fat under pressure; and (3) the presence of open veins which do not collapse.

In bone marrow of adults there is an abundance of fat which, composed principally of olein, is liquid at body temperature. In children fat embolism is rare and this has been explained by Speed,[12] Landois,[13] Zwerg[14] and Timmer[15] by the fact that the bone marrow in children contains relatively little fat and the small amount present is composed of the more viscous palmitin and stearin.

The marrow held in a rigid bony enclosure can be placed under great pressure from hemorrhage and accumulated exudate. The veins supported by bone are prone, when severed, to remain open, thus able to receive the liquid fat under pressure.

If fat enters the blood stream in the majority of cases in which bone is injured one may well ask why symptoms of fat embolism are observed so infrequently. An explanation may be found in the results of the studies of Lehman and Moore,[16] who found that they could inject intravenously into a dog as much as 120 c.c. of cotton-seed oil without producing a lethal result. Since the femur in man contains approximately 65 c.c. of fat, it would appear by deduction that the quantity which enters the blood stream in most cases of fracture would be well tolerated. In the authors' experiments, reported below, several rabbits received all of the fat contained in a tibia and not one of them showed any evidence of ill effect. Gröndahl has stated that fat embolism is directly accountable for not more than 1 per cent of deaths associated with fractures.

Fat emboli are first filtered out in the pulmonary capillaries. If they are sufficient in amount to produce widespread blockage of these capillaries, cyanosis, dyspnea, pulmonary edema and hemorrhagic infarction occur. If they pass through into the general circulation they may be carried to all tissues in the body but they have little or no effect unless they lodge in the brain or heart. When they occlude cerebral or coronary vessels they produce congestion, thrombosis, hemorrhagic infarction and degeneration, and necrosis. The cardiac signs and symptoms are those of coronary occlusion. The changes in the brain give rise to headache, vomiting, delirium, increased or subnormal temperature, coma and convulsions.

Symptoms may appear within a few hours after the injury but usually do not develop until the third or fourth day. They have a cyclic tendency corresponding presumably to the periodic passage

or showers of emboli. A patient who is convalescing normally becomes restless and apprehensive; the respiratory rate increases, as does the pulse; dyspnea, cyanosis and a productive cough develop;

RABBIT - BONE MARROW FAT SUSPENSION

INTERVAL AFTER INTRAVENOUS INJECTION

FIG. 1.

the blood pressure rises and later falls; the temperature may become subnormal or rise to extreme heights; or the symptoms may be predominantly cerebral, characteristically a delirium similar to delirium tremens.

When the symptoms develop within a few hours after injury or operation it often is difficult or impossible to differentiate fat embolism from shock which may also be present and to. which fat embolism may be a contributing factor. When fat embolism appears days later the condition may be indistinguishable from pneumonia or delirium tremens. Undoubtedly fat embolism is a forerunner of pneumonia in some cases. Suggestive, though not pathognomonic, of fat embolism is the presence of fat in the sputum as demonstrated by Warthin and in the urine as pointed out by Reidel.[17] Fat emboli have been observed in retinal vessels with the ophthalmoscope.

Since there is no specific treatment, and as a matter of fact, no treatment other than supportive and symptomatic, efforts in management should be directed to prevention of the entrance of fat into the blood stream.

Caldwell and Huber[18] have shown experimentally that the presence of a tourniquet during and for some time following operation or manipulation of bone greatly reduces the incidence of and the quantity of fat emboli. Caldwell and Huber also found that the

incidence and extent of fat embolism in their experimental animals
was influenced greatly by the activity of their animals following
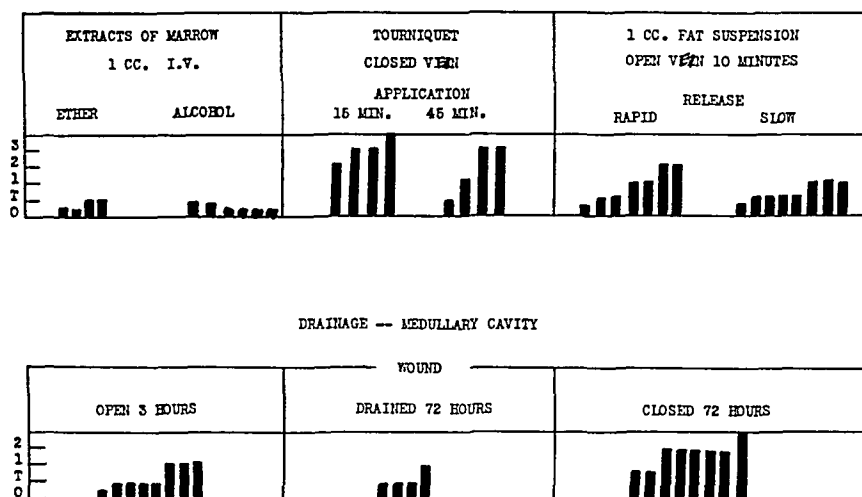operation and therefore recommended immobilization and fixation

EXTRACTS OF MARROW 1 CC. I.V.		TOURNIQUET CLOSED VEIN APPLICATION		1 CC. FAT SUSPENSION OPEN VEIN 10 MINUTES RELEASE	
ETHER	ALCOHOL	15 MIN.	45 MIN.	RAPID	SLOW

DRAINAGE — MEDULLARY CAVITY

	WOUND	
OPEN 3 HOURS	DRAINED 72 HOURS	CLOSED 72 HOURS

FIG. 2.

and liberal sedations. Gauss[19] demonstrated that the presence of fat
increased the viscosity of the blood and the likelihood of thrombosis.
It has been suggested therefore that dilution of the blood with salt
solution was a feasible procedure. Reicket[20] believed that elevation
of an extremity decreased embolization of fat.

In an endeavor to study the influence of certain factors, such as
anesthesia, the tourniquet and drainage, upon fat embolism and to
collect data which might be helpful in lowering the incidence of this
complication in clinical cases the experiments described below were
carried out.

EXPERIMENTAL DATA

Rabbits were used for the experiments and bone marrow fat from
rabbits was used for emboli. The fat was obtained by removing all
bone marrow from both tibias of rabbits used only for this purpose.
The marrow was macerated in a mortar and mixed with salt solution.
It was then drawn into a syringe and injected into other rabbits
intravenously (in one group it was injected intramuscularly). The
anesthetized animals were killed by lethal etherization and those
that had recovered from anesthesia and the controls, or non-anes-
thetized ones, by pithing. The segments of lungs and other tissues
removed for microscopic examination were excised behind hemostats
before the heart ceased beating and were immediately placed in

formalin to fix them before the fat in the capillaries could be washed out with the escape of blood.

All tissue sections were studied and graded. They were graded or classified upon a basis of the quantity of intravascular fat and of the number of capillaries containing fat in a representative member of low power microscopic fields. Gradations ranged from a trace (one or two collections in an entire section) to four (many vessels packed with fat in each low power field). Since an evaluation of the contained fat by this or any other method is at best merely relative, the experimental results must be interpreted accordingly. However, since a high degree of consistency prevailed in the interpretation of specimens obtained from animals subjected to the same experimental procedures, the findings as interpreted have very definite comparative value.

The experiments dealt with varied aspects of the problem of fat embolism and are grouped accordingly.

Group I. The Influence of Fat Solvent Drugs.   Since the few cases of fat embolism observed by one of us (J. D. B.) occurred in patients who had received either no anesthesia or an anesthesia containing no ether and since ether is a fat solvent, the possibility that ether might have a beneficial protective influence in avoiding the catastrophe of fat embolism was suggested. To investigate this possibility equal quantities of bone marrow fat were injected into veins of the ears of two groups of rabbits. One group, after receiving the injections, had the anesthesia continued for varied periods, as outlined below. The other group served as controls, receiving no anesthesia.

1. Specimens recovered five minutes after completion of injections. The etherized animals continued to receive ether during this period and then were killed by increasing anesthesia to a lethal level. The average fat content of the lungs of the twelve etherized rabbits was 1; a trace in five, 1 in four and 2 in three. In eight non-etherized controls, the fat content average was 2.6; 2 in three and 3 in five. Thus there was definitely much less fat in the etherized than in the non-etherized group.

This difference was observed in the first few animals examined and the question of cause suggested itself. Did the ether break down the fat or did it dilate the capillaries so that the fat globules could pass from the pulmonary into the systemic circulation? If the latter were the answer fat should be filtered out in the capillaries of other organs and found in sections of those organs. Consequently in five of the etherized and in four of the non-etherized animals sections were cut from segments of the liver, spleen, kidney, myocardium and

brain. In no instance was fat found in the vessels of any of these organs.

2. Specimens recovered thirty minutes after injection. The average fat content of eight etherized rabbits was 0.85 as contrasted to 2.3 in eight non-etherized. Again there was a striking difference and again no fat was found in the kidneys, spleens, myocardiums, livers or brains of any of the animals.

3. Specimens obtained forty-eight hours after injection. In the animals killed forty-eight hours after they had received 1 c.c. of a fat suspension intravenously the averaged fat content of the lungs was: in the four which were etherized while receiving injections, 2.5; in the four control non-anesthetized, 2.0.

Thus, after forty-eight hours the lungs in both groups contained approximately an equal quantity of fat.

4. Specimens obtained fourteen days after injection, as in (3) with the exception of the time interval between the injection of fat and the recovery of specimens. In the six animals which had been etherized while receiving injections the fat content of the lungs averaged 2.6 while in the five non-anesthetized controls the averaged content was less than 1.0 (0.7). The spleen, kidney, liver, heart and brain of each animal contained no fat emboli.

Curiously, the fat remained unabsorbed longer in the etherized animals.

5. Massive intravenous injections. Each rabbit in this group received intravenously all of the marrow contained in one tibia of a donor rabbit (used only for this purpose). The macerated marrow when injected was suspended in 5 c.c. of salt solution. The lungs were recovered thirty minutes after the injections. The average fat content was 2.7 in the etherized animals and 5.0 in the non-etherized.

As in the case of the animals which received the smaller quantities of fat, a much smaller amount was found in the lungs of the etherized animals but, as would be anticipated, there was much more fat in the lungs of all the animals which received the massive injections. No fat emboli were found in the spleen, liver, kidney, heart or brain of these animals.

6. Injections of bone marrow fat dissolved in 1 c.c. of ether. Since ether is rapidly volatile, considerable difficulty was encountered in making the injections. All animals died while receiving them, presumably of gas embolism. By thoroughly pre-chilling the ether it was possible to complete the injections before the onset of convulsions. In four animals which received complete injections there was no fat in the specimens of lungs examined in two and only traces in two.

7. Injections of bone marrow fat dissolved in 1 c.c. of 95 per cent alcohol. Six rabbits received intravenously bone marrow dissolved in 1 c.c. of alcohol. Three of them were killed twenty minutes later and three after twenty-four hours. No fat emboli were found in four animals and only traces in the lungs of two, both of these after twenty-four hours.

Group II. Influence of Tourniquet.   In this group of experiments a tourniquet was applied to the hind leg of each animal as near the trunk as possible and 0.5 c.c. of the fat suspension injected into an exposed superficial vein. No anesthesia except local infiltration of the skin at the site of exposure of the vein was used.

1. Closed vein. In eight animals the veins remained intact except for the puncture wound made by the needle, and the injected fluid could escape only through the small vessels which were cut in exposing the vein which was injected.

In four the tourniquet was released fifteen minutes after completion of the injections and the animals killed twenty minutes later. The average fat content of the lungs was 3.0. In the remaining four the tourniquet was released after forty-five minutes with the result that the average fat content was only 1.75.

2. Open vein—rapid release of tourniquet. Eight rabbits received injections as in the preceding series, but after the completion of the injections the vein in each animal was opened by dividing it. In the blood which escaped droplets of fat could be seen.

In the four rabbits in which the tourniquet was released after fifteen minutes there was an average fat content in the lungs of 1.25 and in the four in which the tourniquet remained in place for forty-five minutes only 0.75.

3. Open vein—gradual release of tourniquet. The eight rabbits in this series were treated as were those in the preceding series with the exception that the tourniquet was released gradually, permitting rather profuse bleeding to take place for a short period of time.

In all of the animals of this series there were only traces of intravascular fat, and there was no appreciable difference in the quantity present in the lungs of animals subjected to ten or forty-five minutes of vascular occlusion.

Group III. Influence of Drainage of Wound.   In each of the twenty rabbits under local infiltration anesthesia the tibia was exposed and a large hole drilled into the medullary canal. Through the hole a small flexible probe was introduced and the marrow agitated.

In eight animals the hole in the cortex was left open as was the wound; in four it was drained with a Penrose wick and in the other

TABLE I

Group I. Influence of fat solvents.
A. Ether inhaled.
   (a) 5 minutes after intravenous injection of 1 c.c. of fat suspension.
       12 etherized rabbits—T.–5; 1–4; 2–3; av. 1.0.
       8 non-anesthetized—T.–0; 1–0; 2–3; 3–5; av. 2.6.
   (b) 30 minutes after 1 c.c.
       8 etherized rabbits—T.–4; 1–3; 2–1; av. 0.85.
       8 non-anesthetized—T.–1; 1–0; 2–2; 3–4; 4–1; av. 2.3.
   (c) 48 hours after 1 c.c.
       4 etherized rabbits—T–0; 1–0; 2–2; 3–2; av. 2.5.
       4 non-anesthetized—T–0; 1–1; 2–2; 3–1; av. 2.0.
   (d) 14 days after 1 c.c.
       6 etherized rabbits—T–0; 1–0; 2–2; 3–4; av. 2.6.
       5 non-anesthetized—0–1; T–1; 1–3; av. 0.7.
   (e) 5 minutes after 5 c.c. (massive injection).
       6 etherized rabbits—T–0; 1–0; 2–4; 3–1; 4–2; av. 2.7.
       5 non-anesthetized—T–0; 1–0; 2–0; 3–0; 4–0; 5–4; av. 5.0.
   (f) Immediately after intravenous injection of fat dissolved in 1 c.c. of ether.
       4 rabbits—0–2; T.–2; av. 0.25.
B. Alcohol—after intravenous injection of fat dissolved in 1 c.c.
   95 per cent alcohol.
   20 minutes, 0–3.
   24 hours, 0–1; T.–2.
Group II. Influence of Tourniquet.
A. Closed vein, intravenous injection 0.5 c.c. fat suspension.
   (a) 15 minutes after.
       4 rabbits—0–0; 1–0; 2–1; 3–2; 4–1; av. 3.0.
   (b) 45 minutes after.
       4 rabbits—T–1; 1–1; 2–0; 3–2; av. 1.7.
B. Open vein—rapid release—tourniquet
   (a) 10 minutes.
       4 rabbits—0–0; T–1; 1–1; 2–2; av. 1.25.
   (b) 45 minutes.
       4 rabbits—0–1; T–1; 1–1; 2–1; av. 0.75
C. Open vein—gradual release.
   (a) 15 minutes.
       4 rabbits—0–1; T–2; 1–1; av. 0.5.
   (b) 45 minutes.
       4 rabbits—0–0; T–2; 1–2; av. 0.75
Group III. Influence of drainage after trauma to marrow.
A. Open wound.
   (a) After 3 hours.
       4 rabbits—0–2; T–2; av. 0.25.
   (b) After 72 hours—0–0; T–2; 1–2; av. 0.75.
B. Closed wound—bone sealed with bone wax.
   (a) After 3 hours.
       4 rabbits—0–0; T–0; 1–1; 2–3; av. 1.75.
   (b) After 72 hours.
       4 rabbits—0–0; T–0; 1–1; 2–2; 3–1; av. 2.00.
Group IV. Intramuscular injection of 5 c.c. of fat suspension.
   After 48 hours, 5 rabbits—T–2; 1–3; av. 0.8.

eight the hole was sealed with bone wax and the wound tightly closed. In both series half of the animals were killed three hours after operation and half after seventy-two hours.

The average fat content of the lungs of those with drained and open wounds was 0.25 (a trace in three and none in two) in three hours, and 0.75 (a trace in three and 1 in one) in twenty-four hours.

In the sealed series there was definitely more intravascular fat. After three hours there was 1.75 and after twenty-four hours slightly more, 2.00.

Group IV. Influence of Trauma to Bone with Removal of Bone Marrow. The lungs from seven of the donor rabbits or those from which bone marrow had been taken and used for intravenous injections in other animals were examined for the presence of fat emboli. In each rabbit the medullary cavity had been widely exposed and most of the marrow removed. None had received an injection but all had been anesthetized with ether. In four, specimens were obtained thirty minutes after operation and showed no fat in three and a trace in one. Three were killed three days after operation and in the lungs of all three were found traces of intravascular fat.

It is difficult to explain the small number of fat emboli in this series of animals. Probable factors of influence were the extensive removal of marrow, and the free opening of the marrow cavity.

Group V. Intramuscular Injections of Marrow. In five rabbits 5 c.c. of a marrow fat suspension were injected into the muscles of the thigh and the lungs removed forty-eight hours later. Fat was found in phagocytes in the capillaries and in alveoli in all specimens. Quantitatively there was one in three and only a trace in two.

Thus it appears that the liquid fat in the soft tissues was transported to the lungs and presumably transported by means of phagocytes.

Group VI. Normal Rabbits. Since it has been reported that not infrequently the capillaries of the lungs of rabbits normally contain fat, an extensive search was made of the lungs of five normal untreated rabbits and in no specimen was intravascular fat found.

DISCUSSION

The first group of experiments shows that the fat solvents, ether and alcohol, definitely diminished the quantity of fat, demonstrable by fat stains, in the pulmonary capillaries. In the blood within the lungs ether existed in sufficient concentration to have this influence upon the injected fat. The failure to demonstrate fat in the capillaries of tissues in the circuit of the general circulation indicates that the

absence of fat in the pulmonary capillaries is not merely the result of diminished filtration from dilatation of the capillaries by ether. The fact that after elimination of the ether the lungs of the etherized animals contained more fat than the controls seems unreasonably paradoxical. An explanation has been formulated by Lehmann and Moore who obtained similar results in a similar group of experiments. They believe that ether injected or inhaled dissolves and holds the fat until it (ether) is eliminated. The fat therefore is redeposited in the pulmonary capillaries where the ether is given off. Furthermore they have demonstrated that ether, inhaled or injected in a dog after a fatty meal, resulted in the presence of globules of fats in the pulmonary capillaries. This may explain the greater quantity of fat in the etherized animals forty-eight hours after injection. The experiments in group II gave rather conclusive evidence that obstruction of the venous return flow with a tourniquet diminished the number of fat emboli and that washing the vein by releasing the constrictor to a point that reëstablished arterial circulation without releasing venous obstruction further reduced the number of these emboli.

Decompression of the marrow cavity and free drainage diminished the number of emboli. Presumably this prevented the accumulation of fat under pressure.

CONCLUSIONS

The results of these experiments indicate that:

1. Ether anesthesia has little or no beneficial influence upon embolization of fat, and if at all, only during anesthesia or while it is present in the blood stream in moderate concentration. It could have no effect upon the fat which enters the blood stream four or five days after injury or operation.

2. The use of a tourniquet during operations upon bone and the gradual release with some venous bleeding before complete release may lessen the incidence of immediate fat embolism.

3. The prevention of increased pressure by drainage of the medullary canal and wound is probably one of the most important preventative measures. The obvious objection to drainage in clinical cases is the possibility of inducing infection along the drainage tract. As judged from the experience of one of us (J. D. B.) this danger is very remote if drainage is continued not longer than four days.

4. Manipulation of injured bone should be gentle and minimal.

5. Immobilization and elevation reduce the likelihood of emobilization of fat.

6. Fat in the sputum and in the pulmonary capillaries is not pathognomonic of fat embolism. It may arise from liquid fat in the soft tissues transported to the lungs in phagocytes.

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DISCUSSION

ALBERT H. MONTGOMERY (Chicago): We have had the pleasure of listening to a very interesting paper on a relatively rare subject. This matter of fat embolism presents many paradoxical phases. In the first place, pathologists will tell you that about half of the patients going to autopsy will show fat embolism in the lungs. It is well known that many of these cases are unassociated with trauma. The question, then, of the pathologic diagnosis of essential fat embolism must be associated with the clinical phase of the subject. It must be due to trauma for a pathologic clinical recognition of fat embolism to be possible.

It has been interesting to note that in the diagnosis some of the same things hold true, namely, the similarity to shock in the early phases, and the similarity to delirium tremens and alcoholism in the cerebral cases. Although it may occur early, it should not be forgotten that a good basic rule is that given, I believe, by Dennis some years ago, that shock occurs within the first three hours, fat embolism within the first three days, usually between the fifty-sixth and seventy-second hour, and pulmonary embolism about the third week.

There have been many diagnostic factors employed to strengthen this matter. One of them is, of course, the estimation of fat in the blood. It has been found, however, that the average physiologic chemist is unable to give an accurate report that would help to produce a definite conclusion.

Finding fat in the urine is of value, but it must be remembered that fat will float on the urine and that unless the last portion of the urine emptied from the bladder is examined, the urinary examination may be misleading.

In 1877 it was suggested that dark field illumination of the blood might show fat droplets larger than red blood corpuscles. This has been shown experimentally to be of value. Blood removed after the injection of sterile oil has been found by dark field illumination to show large particles of oil in the blood.

More recently Jirka and Scuderi from the County Hospital, Chicago, have shown that as early as twenty minutes after the intravenous injection of oil, x-ray films of the chest will show definite shadowing which they consider as probably indicative of fat embolism.

There is very little to the active treatment, but I think there is considerable to the preventive treatment.

These factors have all been covered by Dr. Bisgard in his paper. Stress should be laid, however, upon the traumatic handling of the bones. In other words, as Lexer pointed out, use sharp electric cutting instruments rather than chisels and mallets in doing this work, whenever possible. The use of a tourniquet appears to me a little questionable, especially as it has been suggested to leave it on about an hour after the completion of the operation. I wonder whether it would not be wiser to do without the tourniquet and risk the chance of a possible fat embolism, seeing that it is fatal in only a few instances.

Experimentally, it has been shown that animals can build up tolerance so that they will take many times the lethal dose of fat. What is it that does this? Possibly some emulsifying agent or perhaps some digestive ferment. Some observers have suggested that work along an experimental line might show some agent of that type that could be used in the treatment of fat embolism, and possibly prolong life where fat gets into the circulation.

Dr. Bisgard deserves considerable credit for this paper. A piece of investigation of this kind will, we may hope, stimulate other work of like nature. We shall probably always have plenty of clinical material on our programs. Research work and investigations of interest to this Society should be encouraged.

Eugene J. Bozsan (New York City): I merely wish to pass on to you the observation of three cases in which I believe fat embolism occurred following the use of high speed electric drill. Two cases were observed after a large-sized drill was driven through the lower end of the femur, and a third when it was used in the neck of the femur. Infarct of the lung occurred, verified clinically and by x-ray examination.

These rapidly spinning large-sized drills have perhaps driven by centrifugal force droplets of fat into the veins, causing the embolism.

This group of cases is, of course, too small for any basic conclusions, but some note may be made of it.

J. Dewey Bisgard (closing): I first wish to thank the discussers for the additions they have made to my paper. In regard to the ratio of

frequency of fat embolism in operated and non-operated cases: I have seen six cases, four of which have been proven, and in the other two the evidence pointed in that direction. Of the six, three were cases that were operated upon, two of them trisacral fusions and one an open reduction. The other three were patients who had had non-operative manipulations. One operated case occurred just four weeks ago, a case in which I had done a trisacral fusion. On the second day the patient's temperature suddenly went up to 106.5, but yet all the clinical signs were inconsistent. She appeared quite well, was rational and her pulse rate was only 100. In other words, the clinical evidence did not check at all with the febrile response, and it appeared that there was a disturbance in the medullary centers regulating heat, presumably upon the basis either of anesthesia with the perivascular changes that go with anoxia or of fat embolism. I think that the symptoms resulted from fat embolism. In three days the temperature returned to normal, and since then the patient has gone along perfectly well.

SIMPLE ANTERIOR DISLOCATION OF THE ELBOW JOINT WITH RUPTURE OF THE BRACHIAL ARTERY

CASE REPORT

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MADISON, WISCONSIN

ANTERIOR dislocation of the elbow joint is an exceedingly rare injury. Simon[1] in 1931, in a review of the literature, stated that Hippocrates first mentioned the condition and added that it may be fatal in a few days. The first case recorded was by Evers in 1787. Simon's own case brought the total then recorded to thirty-one. In 1932, Christopher[2] reported a case. R. Bonn[3] and G. A. Oddone[4] each reported a single case in 1931 and 1932. My own case brings the present number to thirty-five. Included in this list are ten cases where fracture of the olecranon tip was present, a condition not properly included in this series. Excluding these, there are twenty-five cases of true anterior dislocation.

When this rare injury is complicated by rupture of the brachial artery (mine is the only one on record) the surgeon is presented with a combination of injuries that will require his immediate attention and skill, since if not promptly treated the condition may result in gangrene, amputation, and even loss of life.

In the case herewith reported, my nephew, Dr. Reginald H. Jackson, Jr., was present in the operating room with his movie camera and obtained a complete graphic record of the two lesions.

In a series of cases of posterior dislocation of the elbow with vascular injuries reported by Eliason and Brown,[5] the extremity became gangrenous due to the lack of early recognition and institution of surgical care; one patient died, and one case resulted in ischemic contracture.

While Simon in his article on anterior dislocation does not mention rupture of the brachial artery as a complication, he notes that Richet reports one autopsy and that three amputations are reported by Canton,[6] Morel Lavalle, and Rigaud. In all probability, the circulation was interfered with in these cases. This complication, if met today and promptly recognized and treated by immediate surgical interference should give better results. Marnham[7] reports a

successful termination in a case of posterior dislocation where prompt incision with evacuation of the blood clots and ligation of both ends of the divided artery resulted in saving the arm. As in our
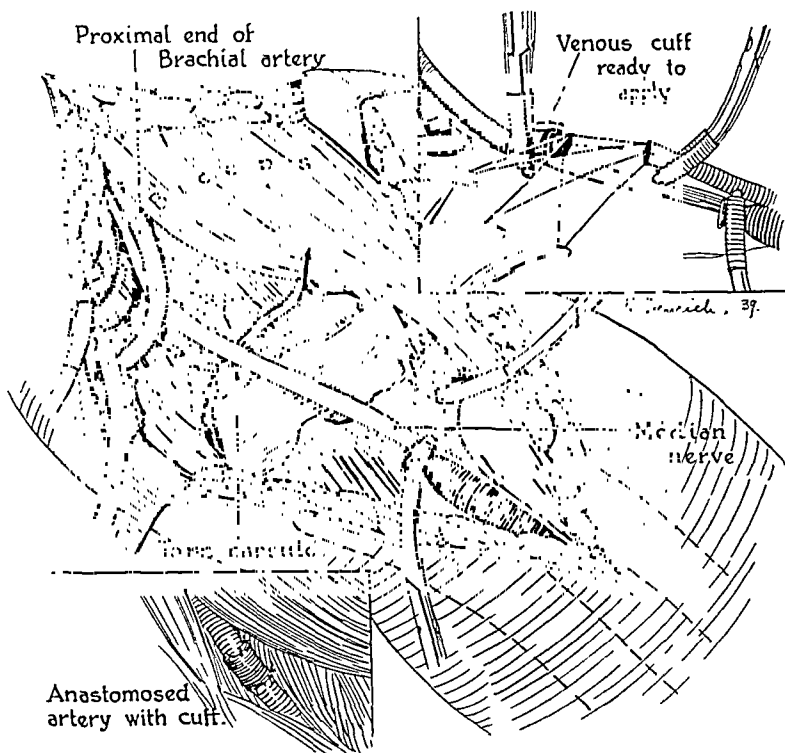

FIG. 1. Shows the ruptured artery and vein, transverse tear of the capsule
of the joint, intact median nerve, intact biceps tendon. Inserts—anasto-
mosing the artery with a venous cuff for reinforcement.

case, the median nerve and biceps tendon were intact. It may be that the anastomosis of the artery in our case was not the determining factor, but it was most encouraging to note the pulsations and the return of color after the continuity of the artery was restored. I have been unable to find a record of any other case in which an attempt was made to anastomose the artery at the time of operation.

On August 2, 1938, E. D., a robust white male, fell a distance of 4 feet, striking on the back of his flexed left elbow. Intense pain and marked swelling with deformity of the joint were present almost immediately following the accident. He was referred to the Jackson Clinic and was seen by me within fifteen minutes of his fall. Swelling in the cubital space was more than one would expect in such a short period of time since injury. The whole forearm was bluish-gray in color and cold. There was only slight sensitivity to pinprick and there was complete absence of the radial pulse.

X-rays revealed an anterior dislocation of the elbow joint which was promptly reduced on the x-ray table, but there was no return of radial pulsation. The patient was taken to his room and pavaex treatment,

FIG. 2. August 2, 1938. Simple anterior dislocation of the elbow joint. Note that there is no fracture of the olecranon connected with this dislocation.

including the arm and forearm, was instituted. Since close observation for a period of two hours revealed no improvement in the circulation, the patient was referred for surgery with a diagnosis of probable rupture of the brachial artery with resulting hematoma and loss of circulation in the extremity.

An anterior incision was made over the elbow joint; after evacuation of about ½ pint of clotted blood, the exact nature of the traumatic lesion was revealed. (Fig. 1.) The anterior portion of the joint capsule had been torn completely across, exposing the glistening cartilaginous lower end of the humerus with the intact median nerve lying directly upon it. Both ends of the torn and separated artery, and also of the torn brachial vein were exposed to view. The proximal end of the artery was pumping a stream of blood. The flexor muscles attached to the internal epicondyle had been severed at their attachment and the brachialis anticus muscle was likewise torn in two. The ends of the artery and vein were clamped, and it then occurred to me that an anastomosis of the artery might be feasible. The clot in the distal end of the artery was removed by suction, and a ½ inch

cuff taken from the vein was slipped over the artery. Anastomosis was performed with interrupted and running sutures of fine Chinese silk, the cuff of vein reinforcing the junction. For a moment there was some balloon-

FIG. 3. November 30, 1938. Show loss of 10 degrees flexion.

FIG. 4. Shows loss of 65 degrees extension; full supination present.

ing and hemorrhage at the point of anastomosis, but with slight pressure of the finger tip, this shortly subsided and soon stopped completely. There followed an almost immediate return of color in the hand and the radial pulse at the wrist was now faintly palpable.
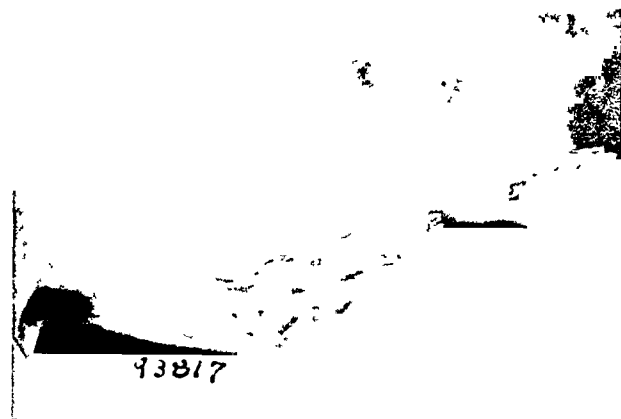

FIG. 5. Shows full pronation.

The joint capsule was closed with interrupted sutures of chromic catgut and the torn muscles were likewise repaired. The field was flushed with saline and the skin united with dermal. Many puncture incisions were made through the skin and fascia of the forearm to allow the serum to exude and prevent a possible ischemic contracture. The arm was dressed and put up in a position of extension with elevation of the extremity.

Eight months following the operation, the patient had a strong radial pulse with no trophic disturbance in the hand, a loss of flexion of 10 degrees and of extension of 65 degrees (Figs. 2, 3, 4 and 5), but was able to do heavy labor without discomfort. Since the roentgenograms show no bony obstruction to motion, the rather large loss of extension is probably due to shortening of the anterior portion of the capsule which occurred in overlapping it in repair. Extension could undoubtedly be increased by a capsulotomy and fascial graft.

CONCLUSIONS

1. Rupture of the brachial artery should always be suspected in injuries of the elbow joint, and especially in dislocations accompanied by absence of the radial pulse and change in color of the hand.

2. Prompt diagnosis with immediate surgical interference is necessary if the extremity is to be saved.

3. Successful anastomosis of the ruptured artery is feasible and greatly lessens the possibility of ensuing gangrene in the extremity.

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DISCUSSION

EDGAR L. GILCREEST (San Francisco): We have had a series of most interesting papers before this Association, but no paper has emphasized more forcibly than this paper of Dr. Jackson's that the object of this Association is to bring back to the fold the well-trained surgeons who may have wandered off into narrow specialties.

The presentation of this case shows so clearly how, in cases of trauma, we need a surgeon who has had broad training. He may not know whether he is going to deal with a fracture, with a joint involvement, or with vascular or nerve surgery.

Dr. Jackson was fortunate in having seen this patient so early. That is most unusual. He is to be congratulated on his early recognition of what

the problem was, on his courage in exploring immediately and, finally, on his ultimate result.

GORDON M. MORRISON (Boston): Cases of this type are as scarce as the proverbial hen's teeth. In ten years at the Boston City Hospital (slightly over 2,000 beds) we have had one lacerated wound of a branchial artery, and that was caused by a sharp implement and not by a dislocation.

It seemed to me that it might be worthwhile to say a word or two about the differential diagnosis that presents itself in a case of this type. Swelling of the arm near or above the elbow, pulse gone, anesthetic cold hand, may be rupture of the brachial artery, or it may be pressure hematoma from a fracture at or near the elbow joint, as described so ably by Stephen Jones two or three years ago.

Still another condition—and it has chanced that I have operated on two such patients—is that of midshaft fracture of the humerus with pressure hematoma so severe that the brachial artery was cut off, giving a picture quite similar to that described so well by Dr. Jackson. The color of arm and hand in his case was bluish-gray. In the two cases of midshaft humerus fracture with pressure hematoma which I saw, the hand was pale and white, as it was in a supracondylar fracture in which I did a fasciotomy. This difference in color is merely of passing interest.

The value of multiple punctures through the skin and fascia, which I think we were all taught ten, fifteen and more years ago, is, to my way of thinking, controversial and of doubtful value in the arm. It would seem that fasciotomy of that part of bicipital fascia called lacertus fibrosus, under which the artery dives, is the one method of attack that really is efficient. However, that is controversial.

CARLETON MATHEWSON, JR. (San Francisco): About two years ago I was walking through one of our emergency hospitals and chanced to look into the operating room to find an alcoholic lying on the table with a dressing over his right arm. The interns explained that he had been picked up off the street about fifteen minutes before, that he had a laceration in the right cubital fossa, that his hand was cold and his radial pulse was missing. The patient was taken to surgery without x-ray examination. Exploration of the wound in the cubital fossa revealed an anterior dislocation of the elbow through a compound wound in the brachial fold. There was a clean transverse laceration in the brachial fold extending down to the exposed lower end of the humerus. Further exploration revealed that the brachial artery had been completely severed and that the anterior portion of the capsule had torn loose from its lowermost attachments. There were no other structures torn. The wound was debrided carefully and we were able to accomplish an immediate end-to-end suture of the brachial artery.

As in Dr. Jackson's case, the radial pulse returned immediately. We made no attempt whatsoever to repair the anterior portion of the capsule, simply reduced the dislocation and put two or three skin sutures in the transverse laceration, dressed the wound, and returned the patient to the

ward. The radial pulse remained palpable for four days. On the fourth day it became very weak and on the fifth day disappeared entirely. On the eleventh postoperative day the pulse in the radial artery returned. During the period the circulation to the hand, which had returned immediately after suture of the artery, remained unimpaired. We believe that the artery became thrombosed after the fourth day and that in the meantime a collateral circulation had developed, sufficient to maintain proper circulation in the extremity. When the patient left the hospital at the end of the third week, he had good circulation in the hand.

Unfortunately, we were unable to follow this patient. We had hoped to obtain an arteriogram to demonstrate what had happened, but the patient signed his release from the hospital at the end of the third week and did not return. Strangely enough, he had almost complete return of flexion and extension in the elbow joint at the time of his discharge.

FREDERICK J. TEES (Montreal): Before I congratulate Dr. Jackson on the splendid results that attended his treatment in this case, I should like to supplement what Dr. Gurd's thanks for the hospitality shown to us, and to say how much we appreciate the opportunity of coming down and joining in the activities of this Association.

It seems rather curious, in view of the great rarity of the condition Dr. Jackson describes, anterior dislocation of the elbow joint, that Dr. McKim, who has been sitting beside me, reminds me of a report that we made ten years ago in which we together described two cases and in which we referred to a prior case which I had published in 1923; Dr. McKim tells me that last year he had a fourth case.

The interest in our cases, in view of what we have heard today, was that in no one of the four cases concerned were there any serious nerve or vessel complications. The difficulty with the first case, though, was that we could not reduce the dislocation. We were led to believe from the textbooks available that the reduction of an anterior dislocation of the elbow joint was the simplest thing in the world. One simply had to put it back; but we worked in vain. Under an anesthetic we all had a try at it, and it refused to go back. Finally, we had to expose it, and what we found was this: that the whole lower end of the humerus had button-holed through a longitudinal slit in the triceps tendon. With the joint exposed it was quite a simple matter to reduce the dislocation and the patient had no further complications.

Dr. McKim's case of last year was identical with that, in that here, too, the end of the humerus had button-holed through the triceps tendon.

ALBERT H. MONTGOMERY (Chicago): In connection with injuries about the elbow joint, it is interesting to consider the possibility of traumatic arterial spasm. I have seen two cases that occurred in supracondylar fractures with closed reduction; when they were put up, the radial pulse was absent, the hand began to get white and then cold. Removal of the restraining apparatus did not restore the circulation, and of course the immediate

thought was that in the manipulation the brachial artery in some way had been injured. In both cases it was cut down upon, but in neither instance was a thrombus found. The artery was not opened, and in both cases, one after one day and the other after two days, the circulation was gradually restored. We were rather glad that we did not try to be such good surgeons, and open them up for resuture.

JAMES A. JACKSON (closing): After these interesting discussions there is little left to be said about the subject. This is a rare condition, but I hardly believe it is so rare as we think. Some of the cases are probably missed.

RUPTURE OF THE SPLEEN

AN ANALYSIS OF TWENTY CASES

JOHN M. FOSTER, JR., M.D. AND DUVAL PREY, M.D.

DENVER, COLORADO

THIS report comprises a review of twenty cases of traumatic injuries to normal spleens, the cases reported being those occurring in four Denver hospitals during the years from 1927 to 1938, inclusive. The authors appreciate the fact that the current and past medical literature contains numerous articles pertaining to this subject, nevertheless a study of ruptured spleens is always a timely one, particularly in view of the increasing incidence of such accidents resulting from our present mechanical age. Statistics indicate that rupture of the liver is a far more common accident than is traumatism to the spleen, but from a purely surgical viewpoint splenic ruptures are more important, because extensive damage to the liver is frequently so rapidly fatal that surgery cannot be attempted. The diagnosis in such cases is determined only at post-mortem examination.

ETIOLOGY

Rupture of the spleen is produced by violence of different kinds, and, furthermore, the type seems to vary somewhat in different communities. For example, Dretzka reported a series of cases in Detroit, the center of the motor industry, in which, surprisingly enough, gunshot wounds were the predominating factor, while in our own series the automobile exacted the highest toll, 15 cases, or 70 per cent. Of the remaining six cases, five followed falls and one was caused by a stab wound.

Conforming with other statistics, there was a preponderance of males in the proportion of 9:1. The average age of the victims was 20 years, the oldest being 43 and the youngest 5 years of age. In this series almost one-half of the patients were younger than 15, which bears out Connor's contention that the recklessness of youth and the inability of youngsters to care for themselves in the presence of danger is a very definite contributory factor in the etiology of this accident.

SYMPTOMS AND SIGNS

The symptoms and signs in splenic ruptures have been so often and so accurately described that any review must seem superfluous,

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but the fact remains that only too frequently surgery is delayed unnecessarily long because of failure to arrive at a correct diagnosis. In the present series of cases it was discovered that several of the patients were explored with an indefinite preoperative diagnosis of "possible or suspected rupture of a viscus."

Of the symptoms abdominal pain was, of course, outstanding, having been the admitting and chief complaint in every instance. The character of the pain varied in different individuals, but for the most part was described as generalized and in only 30 per cent of the cases was the discomfort localized to the left half of the abdomen. Pain referred to the left shoulder (Kehr's sign), described so frequently, was remarkable by its absence. The next most constant finding was tenderness which was found to be present in all save one case, but similarly was not localized in the majority.

TABLE I

SYMPTOMS AND SIGNS IN ORDER OF FREQUENCY

	No. of Cases	Percentage
Pain	20	100
Pain localized	6	30
Tenderness	19	95
Rigidity	17	85
Localized to L.U.Q	3	15
Shock	13	65
Dullness	10	50
Vomiting	7	35
Distention	3	15

Shock, of a greater or lesser intensity, should be an almost constant finding in any case of splenic rupture with the exception of those rare cases of delayed hemorrhage. In our review, rather profound shock existed in thirteen, or 65 per cent of the cases, and when present was an important aid in diagnosis. Syncope or unconsciousness was described in only a single instance. This, we believe, is a vastly overemphasized sign, in spite of Quenu's belief that syncope is pathognomonic of splenic rupture if delayed.

Regarding the importance of vomiting as a symptom, there is a considerable difference of opinion—some authors believing that this symptom is so common in most intra-abdominal diseases that its presence is not particularly significant; but on the other hand certain men (Connor, Robitshek, Trendelenburg, and others) feel that vomiting occurs frequently enough in rupture of the spleen to be a sign of some importance. The average incidence in various

series of case reports seems to be approximately 20 to 25 per cent, while our figures were somewhat higher; i.e., vomiting occurred in seven of the twenty cases (35 per cent).

Rigidity, generalized or limited, was encountered in eighteen of the twenty cases, but unfortunately from the point of view of diagnosis, in only three cases was the rigidity localized to the left upper quadrant. Dullness, a sign of equal or greater importance was described in 10, or 50 per cent, of the cases, while shifting dullness was elicited in only four. In our opinion, attempts to determine the presence or absence of dullness in the flank have been sadly neglected, for there can be no doubt that such a positive finding is the most important single clue to correct diagnosis and early intervention.

White blood counts preoperatively were done in fourteen instances, the average count being 14,500 cells per cu. mm., which conforms quite well with reports from other clinics. It is our feeling that the white blood count is of relatively little diagnostic value unless in excess of 20,000, when, under such circumstances, hemorrhage may be suspected. Red cell counts were done so infrequently that no statement can be made regarding their value.

The presence of associated injuries in this analysis did not present the usual grave complications so frequently described, and in only one case (rupture of the spleen with rupture of the left lobe of the liver) could the fatal outcome be even in part attributed to other injuries. However, in addition to the above case, the following complicating injuries were noted: fracture of ribs (three cases); penetrating wound of the liver (one case); laceration of the diaphragm (one case); and contusion of the left kidney (one case).

DELAYED HEMORRHAGE

Delayed hemorrhage, an unusual but extremely dangerous complication of splenic traumatism, has received but scant attention in the past, and our present better understanding of this subject has been largely made possible by the splendid contribution of McIndoe, who, in 1932, collected and carefully reviewed forty cases of secondary hemorrhage following injuries to the spleen. Gardiner, in 1935, reported an additional case and remarked that there were fewer than fifty such cases in the literature, and since that time there have been only rare further case reports.

Despite the obvious infrequency of delayed hemorrhage, a thorough appreciation of its significance and a suspicion of its

possible presence must be constantly borne in mind, because the secondary hemorrhage is frequently so rapid in its onset and so copious in amount that surgery is unavailing. In support of this we find that McIndoe in his review states that there were seven patients who died of secondary hemorrhage before operative intervention could be attempted.

Delayed hemorrhage is not to be confused with a slowly progressive hemorrhage from a small tear in the spleen. In the former, there must be a definite latent period or a so-called period of "symptomatic silence." Arbitrarily, delayed hemorrhage is spoken of as such only when this latent period is in excess of forty-eight hours —statistics indicate that secondary bleeding occurs most often between the third and seventh days.

Some worthwhile facts are obtained from a study of the reported cases of delayed hemorrhage. In the first place, the initial trauma is usually of a less severe nature, which in part accounts for a certain lack of careful observation during the latent period. McIndoe and others believe that there are certain clues to diagnosis during this asymptomatic interval. The two most important diagnostic signs, according to these authors, are the persistence of dull, remittent left upper quadrant pain, and slight localized rigidity. It is of interest that secondary trauma was not a causative factor in the initiation of the delayed hemorrhage, for most cases occurred with the patient at rest or after slight straining, such as might occur from bending over or during the act of defecation. Furthermore, when the secondary hemorrhage started, in the majority of the cases its onset was very abrupt and immediately followed by the classical signs associated with primary rupture of the spleen, namely, severe pain, rigidity, tenderness and shock, and thus the indications for early operation were perfectly apparent.

Numerous authors (Neck, Demoulin, Nast-Kolb, Jackson) have attempted to explain the pathology in the spleen which might account for delayed hemorrhage, but the most satisfactory summary is again obtained from McIndoe's review. Apparently in one-half of the cases the secondary hemorrhage was due to rupture of a subcapsular or intrasplenic hematoma, while the remaining 50 per cent were found to have a walled off perisplenic hematoma which for one reason or another suddenly ruptured into the general peritoneal cavity. The author further calls attention to the anatomic structures in the left upper quadrant which help nature wall off a perisplenic hematoma, the ones of chief importance being: the diaphragm above; the splenic flexure below; the stomach, intestines,

and gastrocolic ligament to the right; the parietal wall laterally and behind; and the omentum and anterior abdominal wall in front.

In the present series, one of the authors (J. M. F.) had occasion to treat one case of delayed hemorrhage (three days) in which the patient recovered after a stormy convalescence following surgery. Because of the scarcity of such cases, it seems warrantable to report this one in some detail.

A boy of 13, while running, tripped over a low wire fence, fell forward and struck his chest and abdomen. He was transported to the Denver General Hospital and x-rays of the chest, were taken. Since these were negative, he was released and allowed to go home. He remained at home the same day and the next, the only complaint being mild colicky pain in the left upper quadrant.

He was first examined by me at his home thirty-six hours after the accident and at this time did not appear to be acutely ill. The temperature was 98.4°F., the pulse rate 68. The lungs were clear throughout and examination of the abdomen revealed only slight tenderness in the left upper quadrant. There was definitely no spasm, rigidity, or dullness in the flank. The following afternoon the pain recurred and became gradually worse during the night, and early the next morning he vomited coffee ground material. At the time of my examination at 9:00 A.M. the patient was obviously suffering a great deal, the temperature was 99.6°F. and the pulse rate 85. There was marked rigidity and dullness of the left side of the abdomen. The hemoglobin was 80 per cent, red blood cells 3,500,000 per cu. mm., and white count 12,500, polymorphonuclear cells 92 per cent. Rupture of the spleen with delayed hemorrhage was diagnosed and immediate operation was advised and accepted.

Through a left rectus incision more than a pint of blood was evacuated. The bleeding originated from a laceration of the superior pole of the spleen and could be nicely controlled by pressure. Therefore two gauze packs were inserted to this area and the abdomen closed in layers, using double suture material and six silkworm gut retention sutures.

On the eighth postoperative day the two gauze packs were removed without difficulty and without hemorrhage, but twelve hours later the boy complained of pain in the wound, his pulse became more rapid, and he vomited once. Inspection of the wound revealed fresh blood on the dressing and a diagnosis of secondary hemorrhage from the spleen was made. At operation there was no evidence of splenic hemorrhage; the bleeding was due entirely to wound disruption which was repaired without drainage, using interrupted heavy silk sutures through all layers.

The immediate convalescence, with the aid of a small transfusion, was essentially uneventful for a further period of eleven days. Then the patient developed symptoms of partial obstruction which was confirmed by x-ray. A third operation through a right rectus incision disclosed a mass of matted

together coils of small intestines which were carefully freed. Due to the poor condition of the patient a resection was considered inadvisable, and a jejunostomy was therefore done through a stab wound incision and the abdomen closed in layers without drainage.

Following this last procedure the drainage from the jejunostomy opening was profuse but finally ceased. Five weeks postoperatively the patient was discharged from the hospital. He has been carefully followed for a period of nine years, has been entirely well, and has shown no evidence of recurrence of obstructive symptoms.

<div style="text-align:center">TREATMENT</div>

The foremost consideration in the treatment of splenic rupture is to determine the proper time to operate. One of the major errors seems to be difficulty in arriving at the correct diagnosis early, which may result in a serious delay and materially reduce the individual's chance of survival. In our series, the shortest interval between admission and operation was one hour, the longest sixty hours, while the average interval was twelve hours. However, in those cases terminating fatally, the average interval was only 7.5 hours, so early operation is by no means the entire answer. Certainly surgery should not be contemplated upon the patients evidencing signs of severe systemic shock, for in this type of case, careful preoperative preparation to combat the shock is essential.

The use of transfusions prior to surgery is of vital importance and is a greatly neglected adjunct to the shock treatment. Most authors extol the merits of transfusion, but in almost every instance its use is reserved for the postoperative phase. In the six fatal cases of this review, preoperative transfusion was instituted in only one patient and certainly there was ample time before operation (average of 7.5 hours) to have secured a suitable donor. Furthermore in the remaining fourteen favorable cases, preoperative transfusions were given in but three instances. To go a step further regarding the infusion of blood this seems a fitting place to call attention to the invaluable benefits of autotransfusion during surgery. It has only been comparatively recently that this life saving maneuver has begun to receive its proper recognition in this country, although Theis in Germany first suggested its advantages in 1914. Downing and Larsen in 1934 reviewed the literature pertaining to this subject and concluded that autotransfusion was a perfectly safe procedure, and since that time sporadic case reports (Reuton, Kraft and others) have appeared in which retransfusion in splenic rupture was accomplished without incident and with material benefit. In this series autotransfusion was carried out in only two instances—in

both patients with success and with a favorable outcome, due in a large part, we feel sure, to this procedure. Unfortunately, autotransfusion was not attempted in any of the fatal cases.

The type of surgery to be used depends to a certain extent upon the individual case. We are not in accord with certain authors who are so emphatic in stating that splenectomy is the sole method of treatment, although we are inclined to agree with McIndoe that removal of the spleen is the only method applicable in cases of delayed hemorrhage.

In the vast majority of cases splenectomy remains the procedure of choice for several reasons, among which may be mentioned: (1) the bleeding is immediately controlled and with less chance of secondary hemorrhage, (2) there are distinctly fewer postoperative intra-abdominal complications, and (3) it is the only method applicable in ruptures involving the large vessels at the hilus. Removal of the spleen was performed in twelve of our twenty cases, with four deaths, a mortality of 33.3 per cent. (Table II.)

TABLE II
OPERATIVE PROCEDURES

Type of Operation	Lived	Died	Per Cent Mortality
Splenectomy	8	4	33.3
Suture	2	1	33.3
Tamponade	3	1	25.0
Suture and tamponade	1	0	0
Total	14	6	30.0

Splenorrhaphy or suture of the spleen has only a limited field of usefulness and is to be used only in selected cases with small tears, due to the friability of the splenic tissue and the consequent difficulty of obtaining thorough hemostasis. This method was successful in two of the three cases in which it was attempted (Table II) in this series. Although most authorities condemn this type of operation, Dretzka is more enthusiastic and in 1930 reported ten patients in whom suture or suture and tamponade was accomplished with only two deaths, but it is of interest to note that more than 50 per cent of his cases were due to gunshot or stab wounds, such lacerations naturally favoring splenorrhaphy.

It is evident from a study of various case reports that tamponade has been used somewhat extensively in the past and some authors

(notably Barnes, Wallace, Rose, and Brogstitter) have gone so far as to state that it is almost the ideal treatment in ruptures, with the exception of those of the hilus. On the other hand, however, there are four tenable objections to the treatment by packing: (1) the spleen is too mobile successfully to pack a bleeding part; (2) the removal of the pack frequently will produce further hemorrhage; (3) the risk of infection; and (4) the danger of adhesions is materially increased.

In spite of the fact that tamponade was performed in four cases in this report (Table 11) with only one fatality, it is our feeling that there is only a single indication for this procedure, namely, in the desperate risk patient in whom the general condition is so critical that it is obvious to the surgeon that any attempts at extirpation of the spleen would spell disaster. Under such circumstances there are a few cases saved by tamponade which otherwise would undoubtedly terminate fatally. Such a case was seen by the authors. Very briefly the history is as follows:

The patient, a man of 30, was attacked in September, 1928 by an unknown assailant with a knife. He immediately collapsed and was transported to the Denver General Hospital, where he was seen within an hour after the accident. Physical examination revealed a well nourished man in extreme shock, with ashen gray facies, subnormal temperature and extremely rapid, weak pulse. The abdominal examination showed an incised wound 7 inches in length, extending transversely beneath the left costal margin from the epigastrium to the tip of the eleventh rib. Almost the entire stomach, which did not seem to be injured, had herniated through the wound.

The man was immediately transferred to the operating room, where a saline infusion was started, and he was prepared for operation. After reduction of the herniation of the stomach, more than a liter of fresh blood escaped from the wound. The stomach, transverse colon, splenic flexure, and left kidney showed no evidence of damage, but there was a good sized rent in the lower pole of the spleen complicated by a laceration extending through the diaphragm into the left pleural cavity. Since the patient was pulseless, the idea of splenectomy was abandoned, and two 5 yard rolls of gauze were inserted as packing into the left hypochondrium, the free ends being brought out at the lateral angle of the wound. Six interrupted heavy silk sutures were inserted through all layers to approximate the wound edges. The immediate condition for several days was very critical due to a complicating left-sided pneumonia of traumatic origin, but after ten days, the packing was gradually removed, a little each day, following which recovery was uneventful.

COMPLICATIONS

The two most common complications in this series were wound disruption and intestinal obstruction. (Table III.) Evisceration has occurred so frequently following operations for ruptured spleens that Bailey suggests that this complication may be due to digestion of the suture material by pancreatic ferments. Whether or not this theory is correct, the fact remains that disruptions are frequent enough to consider replacing the usual wound repair with the use of interrupted braided silk sutures through all layers, a procedure which has been extremely satisfactory in our hands and has the twofold advantage of preventing disruptions and also of saving operating time at the conclusion of the procedure when a few minutes may mean the difference between success or failure. Of the two patients in this report showing evisceration, one recovered following resuturing but only after a stormy convalescence, while the other, with a disruption on the tenth postoperative day, subsequently died of peritonitis on the fourteenth day.

TABLE III
COMPLICATIONS

Nature of Complication	No. of cases	Died
Intestinal obstruction	2	1
Evisceration .	2	1
Mastoiditis	1	0
Empyema	1	0
Total	6	2

Intestinal obstruction likewise was a complication in two patients, and again with one recovery and one death. As stated previously, we believe that intra-abdominal adhesions and obstructive symptoms occur more frequently following tamponade, although in this series the obstruction followed tamponade in one case and splenectomy in the second, with the fatality in the latter case.

Because of the close relationship of the spleen and the left lower lobe of the lung, pneumonia or pneumonia with empyema has been one of the more common complications encountered in the literature, but in the present report there was but a single instance in which this complication was present.

SUMMARY

1. A detailed analysis is made of twenty operated cases of splenic rupture observed in four Denver hospitals during the years from 1927 to 1938, inclusive.

2. It is felt that the correct diagnosis is not made frequently enough and, more particularly, not early enough. The two especially diagnostic signs of rupture of the spleen are localized abdominal rigidity and shifting dullness.

3. Associated injuries in this review played only a small part, and in our opinion there was but a single fatality attributable to injury other than that to the spleen.

4. The importance of delayed hemorrhage following splenic traumatism is recognized in spite of the infrequency of its occurrence. The etiology, pathology, symptomatology and treatment of secondary hemorrhage is discussed and a single case observed by one of the authors is reported in detail.

5. Preoperative transfusion and autotransfusion are life saving procedures which have not received proper recognition in our own series of cases or in those of others.

6. The treatment of choice in the vast majority of cases is splenectomy. Suture alone is indicated only in rare, selected cases with small lacerations, such as occur following gunshot or stab wounds. Tamponade, we feel, should be resorted to only in the desperate risk case in whom any other type of surgery would undoubtedly cause an operating room death.

7. Because evisceration is one of the most common postoperative complications, it is suggested that the usual wound repair be replaced with the use of interrupted heavy silk sutures through all layers.

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DISCUSSION

ROSCOE C. WEBB (Minneapolis): I have enjoyed Dr. Foster's presentation and have been very much interested in his contribution. There are a few points which I would like to discuss.

In connection with diagnosis we frequently read about Ballence's sign, referring to the increased splenic dullness due to the accumulation of blood clots about the splenic area. In this same connection an x-ray may be very useful. The x-ray may not only show cloudiness in the region of the spleen, but also, as in a recent case of my own, elevation of the diaphragm on the left side and displacement of the stomach toward the right side. The x-ray might also reveal a rib injury.

Dr. Foster referred to the frequency of injuries of the spleen occurring in younger patients. It is quite probable that this is due to the more elastic overlying chest wall of children.

I should like to bring up the question of spontaneous recovery of injured spleens for the consideration of this organization. In the numerous papers on ruptured spleens the cases are frequently divided into four groups: first, those who rapidly succumb and never rally; second, those who recover from the initial shock and present signs of a ruptured spleen; third, those who present signs of delayed hemorrhage two or more days following the initial injury; fourth, patients who are diagnosed as having ruptured spleens but recover spontaneously. I wonder if any of them ever recover spontaneously or if we are justified in considering spontaneous recovery as possible. As long as spontaneous recovery is unqualifiedly listed in the published articles we are apt to be led toward conservatism, and conservatism in the presence of rupture of the spleen is apt to be disastrous. Turnbull of London is quoted as saying that he never had seen at necropsy a spleen containing the scar of an old injury. James S. McCartney recently reviewed 25,000 autopsy records performed by the University of Minnesota Department of Pathology from 1920 through 1937 and found no instances of healed splenic hematomas. E. T. Bell's experience has been the same. It would therefore appear that spontaneous recovery should only be mentioned with qualifying remarks.

The cases of splenic rupture with delayed hemorrhage are very rare, as Dr. Foster has mentioned. I would like to mention the case of an 8 year old boy who was struck by a puck while watching a hockey game in February, 1939. He complained of pain and was carried home by his father. He was up and about the next day and did not present severe symptoms until the fourth day when he was leaving school. While putting on his coat he had an attack of pain in his left side and he vomited. I saw him one week after injury. His symptoms and signs were classical and on splenectomy the spleen was found to be lacerated at the junction of the upper and middle thirds on the outer surface, and there were two subcapsular hematomas. A transfusion of 300 c. c. of blood was given both before and after the splenectomy. Recovery was uneventful.

HERBERT H. DAVIS (Omaha): Dr. Foster has presented an excellent review of a fairly uncommon lesion. My discussion is based upon the review of histories of eleven cases of ruptured spleen, five from the University of Nebraska Hospital and six from various doctors in Omaha.

As Dr. Foster reported, the automobile accident is a fairly common cause. Four out of our eleven were due to this and all the mortalities fell in this group. Two were the result of coasting accidents, the patients being brought into the hospital within a few minutes of each other. One was in a youngster with hemolytic icterus. His mother, one of his brothers and a sister had had splenectomy and another brother is planning upon having a

splenectomy this year. Probably the hemolytic icterus has something to do with making that spleen more liable to rupture.

Eight of our patients were males and three females.

Dr. Foster did not find left shoulder pain in the records of any of his cases. We would expect it to be fairly common because any irritation of the central portion of the diaphragm is likely, reflexly through the phrenic and supraclavicular nerves, to cause this symptom. In our cases, six of the eleven had definite left shoulder pain. In only one was it stated that there was no pain. In the other four it was not mentioned. I think if the people who had taken the histories had been on the lookout for this, we probably would have found more cases with that symptom.

Fullness in the flank is also a symptom, especially when there is a very severe hemorrhage, and should certainly be looked for. Percussion of the spleen will often give us a clue that we would not otherwise get.

Six of our cases had complete rupture of the spleen with severe intra-abdominal hemorrhage. A subcapsular or localized hemorrhage was in four, and delayed rupture, with delayed hemorrhage in one.

This latter case may be reported in a little more detail. A woman, age 38, received in an automobile accident a blow over the left chest which was followed by pain in the left upper quadrant of the abdomen and left shoulder. She vomited once. She did not see a doctor until the next day. He strapped her chest, thinking she had a thoracic lesion. The following day she went back to her work in a laundry and worked right along until the fourth day after the accident when at ten in the morning she had sudden severe pain in her left upper quadrant and epigastrium which soon became generalized. Nausea and collapse ensued. That evening she was taken to the University Hospital, where the chief findings were her pallor, air hunger, marked generalized abdominal pain and tenderness, inaudible peristalsis, and dullness in both flanks. The pulse was 132, blood pressure 118/78, hemoglobin 25, and white count 12,600. She was given a blood transfusion and operated on. At operation the spleen was found to be literally blown to pieces. Part of it was clear down in the pelvis. That which was left was just mushy, and no real splenic tissue could be made out. The hemorrhage was so severe that the surgeon packed the wound, and stopped the hemorrhage in that way, but the patient died ten hours later.

The suggestion comes to my mind that if these cases have symptoms of splenic lesion, it might be a good plan to keep them in bed to lessen the possibility of delayed hemorrhage.

WARREN H. COLE (Chicago): My experience with splenic rupture has been limited to a few cases, but I do wish to emphasize one or two points. One of them is the difficulty one frequently encounters in attempting to suture lacerations of the spleen. The spleen commonly is so mushy that sutures will not hold; attempts to apply sutures usually result in more

hemorrhage. I think it is well worthwhile not to waste time trying to suture these lacerations if it appears obvious that splenectomy may be quite simple. Splenectomy in patients with spleens that are normal except for the laceration, usually is quite easy. The only real contraindication is the serious condition of the patient. Moreover, these spleens are occasionally so badly pulverized that it is not worthwhile to try to save them, not nearly so necessary as it would be in laceration of the kidney where function is necessary for life.

I am also impressed with the danger of postoperative hemorrhages; a further etiologic factor is that, following trauma, thrombosis of the various veins may occur and progress to such a degree as to occlude a major trunk of the splenic vein. This may lead to a very marked edema and congestion of the spleen. From experimental and clinical findings we know that rupture of the spleen may then occur spontaneously.

I wish also to emphasize the use of autotransfusions. I know it is not used enough. True enough, one may be able to find a donor rapidly, but all of us will admit that the blood of patients when spilled in the peritoneal cavity should not be wasted; if a few hundred c.c. can be salvaged, it should be put into sodium citrate solution and used as a transfusion. I have resorted to this procedure in two instances. In one of these patients there were a lot of clots in the peritoneal cavity. It has been said that autotransfusion of blood salvaged from partially clotted blood is associated with a danger of severe reactions. However, I have heard of the use of such blood on several other cases, but in no instance, including my own, did a reaction occur. If an intestine has been perforated in addition to the splenic hemorrhage, the blood may obviously be contaminated, and should then be used only in dire emergencies.

KELLOGG SPEED (Chicago): I promised Dr. Foster to discuss his very excellent paper. May I modestly report seven cases of ruptured spleen in which I have operated. Three were gunshot wounds, two were coasting accidents, and the others were falls from a height.

I merely wish to touch upon a few practical points. Where the patient is in shock and there is undoubtedly a hemoperitoneum, one may be in doubt what organ is involved. You may not be able to find by palpation in the flank that the kidney has been injured, but in two of my cases the liver and the kidney on the opposite side were ruptured, and in one case the kidney on the same side was ruptured.

In one case I opened the abdomen, and finding some of the blood clotted, I did a Heller's test with nitric acid, believing that if there were free bile from the ruptured liver, I would have a key as to the exact location of the principal rupture. That helped me because there was no bile, and I was able to devote my attention purely to the spleen.

When the spleen is ruptured, being the graveyard of red cells, it possibly furnishes all the materials for an early and complete clotting. When hemoperitoneum and shock appear late it is evidence of a bursting of the

clot or the wall of the clot, whereas blood from the liver containing the taurocholic or glycocholic acids and their salts is inhibited from coagulating, and one is very liable to find a mass of dark, thick blood without any coagulating at all.

In one patient, the gunshot wound was not directly over the spleen, but perforated the chest wall, went through the edge or back surface of the transverse colon, and then struck the spleen. The piece of shrapnel—this was during the war—produced a splitting result. The missile went clear through the organ, and the radiating splits in the spleen opened up, leading to serious and in this case nearly fatal hemorrhage.

It is very difficult to expect a tamponade to cure that sort of rupture.

JOHN M. FOSTER (closing): I have very little, if anything, to say in closing. I am grateful to the gentlemen for their discussion. I might say that a possible reason we did not have some of the signs ordinarily present, is that the majority of these cases were observed in one of the city hospitals. The histories are frequently indifferent, and perhaps some of the signs and symptoms have been overlooked.

In delayed hemorrhage cases, it is most remarkable how a spleen can be found, part of it in the right half of the abdomen and yet have no hemorrhage occur for several days. Statistically, I believe the average length of delay in these cases is from four and seven days, which is a surprisingly long interval.

PRIMARY REPAIR OF SEVERED TENDONS

THE USE OF STAINLESS STEEL WIRE

STERLING BUNNELL, M.D.

SAN FRANCISCO, CALIFORNIA

OF the many tendons throughout the body, those which are most frequently severed are in the hand and wrist and in the foot and ankle. The commonest sites by far are in the hand and wrist, the most frequent of all being the flexors in the proximal segment of a finger.

The large tendons, such as Achilles, hamstrings, quadriceps, biceps and triceps, are not difficult to repair successfully, but those frequently severed and presenting a more difficult problem are about the hand and wrist and the foot and ankle where the tendons pass through narrow rigid tunnels. Here, after tendon repair, adhesions to sheaths too often limit or prevent movement. If loose movable tissues surround the repaired tendon, success is almost assured, but not so if the repair is in firm synovial lined fascial tunnels. Thus extensor tendons give the best results, then come flexors in the forearm and in the palm, and worst of all, and also the commonest, flexors in the proximal segment of a finger. Here the tunnel made by the annular ligament is firm, narrow and over an inch long. When the problem of repair here is solved, the repair of all other tendons is made possible.

PRIMARY TREATMENT OF THE WOUND

Before specifically discussing primary tendon repair let us first lay the foundation of first aid treatment of wounds in general.

The proper early surgical procedure determines the fate of the patient, in that if tendons, bones, and joints are injured and exposed the patient can, by the first treatment, be spared long illness from sloughing tendons, infected joints and osteomyelitis and will also be spared extensive permanent crippling. This can be done by operating early, debriding thoroughly and by saving all vulnerable tissues by covering them over.

1. *Time Factor.* All wounds are potentially infected. By debridement these germs can be excised. The golden time to operate, therefore, is soon after the accident and before the germs have had time to multiply. We can then, by debridement, convert a laceration into a clean surgical wound and thereafter reconstruct the nerves, ten-

dons, bones, etc., and cover over all vulnerable tissues. Also we can
use local, block, brachial plexus or spinal anesthesias, which are
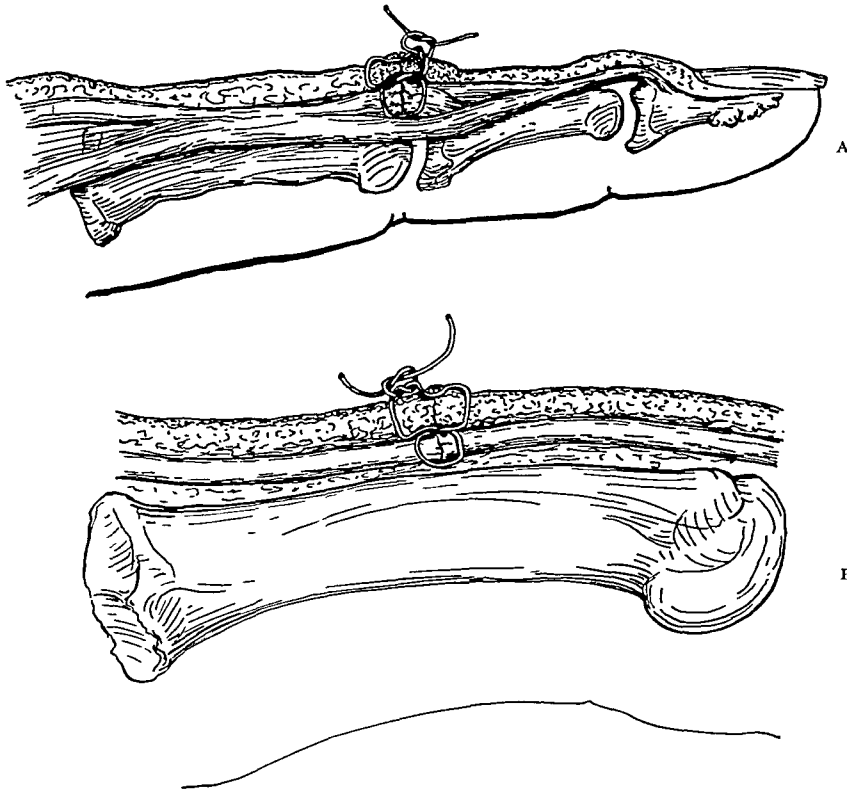dangerous in the presence of infection.

Fig. 1. A, primary suture of severed central slip of extensor tendon with
   simple figure of eight stainless steel wire relying more on splinting to pre-
   vent parting. B, similar method of primary suture of tendon on dorsum of
   hand.

Many authors place the limit of time for suturing tendons at from
two to six hours following the accident. They base this on hospital
statistics of operations done over many years and by multiple
surgeons and do not stress the value of debridement and splinting. It
is my conviction that, given a clean-cut laceration which has not
been tampered with, one can, by thorough debridement, followed by
postoperative immobilization, elevation and dry air dressing, suture
tendons up to twenty-four hours after injury. Primary union can be
obtained providing that a smear is free from cocci and excess of pus
cells. Of course, it is preferable to suture tendons under twelve hours
and the earlier the better, but if we observe all other factors we can
in selected wounds succeed up to twenty-four hours.

Beyond twenty-four hours tendons should never be primarily su-
tured and even within twenty-fours hours only under the best of

conditions. Tendons should not be sutured even early, in the presence of extensive contusion, lowered vitality of tissues and ground in dirt. Contamination from out of doors is worse than that received indoors.
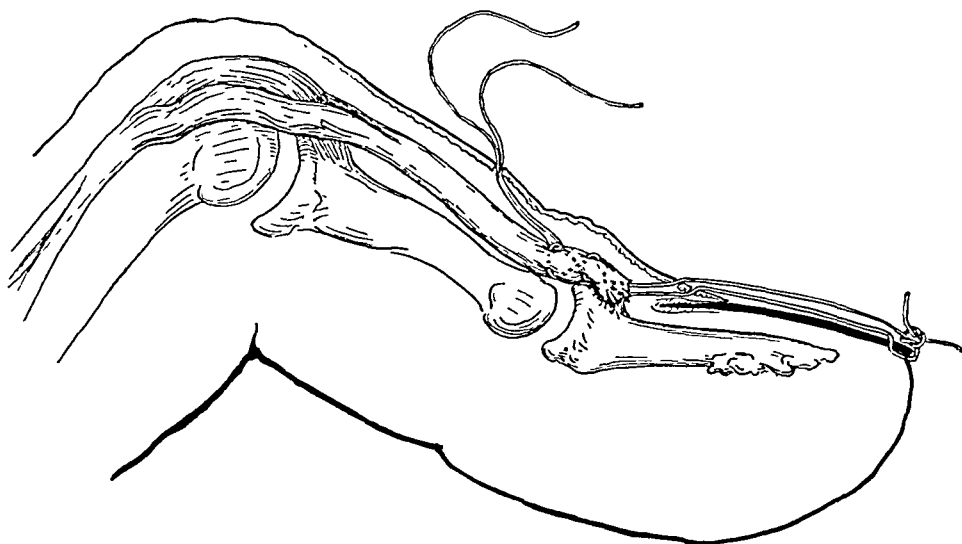

FIG. 2. Method of using stainless steel wire for late repair of insertion of extensor tenson, fastening the suture wire to the finger nail and later removing it by the pull out wire.

Tendons should not be sutured primarily unless hospital facilities are good and only by a surgeon trained in this particular field.

2. *Debridement.* The second important principle to observe in the primary suture of tendons is to do a careful and thorough debridement. Without this, tendons should not be sutured as the wound is destined to go through a stage of ridding itself of a layer of traumatized infected tissue by necrosis, sloughing and infection. Infection frequently follows such tendon suture and is often extremely severe. The following is the technique referred to by the term debridement.

Cover the wound with gauze and shave and cleanse the surrounding area with soap and water. With gauze still over the wound, wrap the limb in a towel and wind it bloodless from the fingers to above the elbow with a rubber Esmarch bandage. One and one-half inches above this apply the cuff of a blood pressure apparatus and pump to 300 mm. Hg., clamp the tubes and remove the Esmarch. An Esmarch tourniquet should never be used as it causes paralysis.

Paint the surrounding area with equal parts of tincture of iodine and alcohol. After giving local or block anesthesia (never adrenalin in digits) iodize the wound in all its depths.

Debride the wound, that is, convert it into a nontraumatized aseptic wound by excising its complete surface, sparing essential structures as nerves, vessels and tendons. With sharp, flat, curved
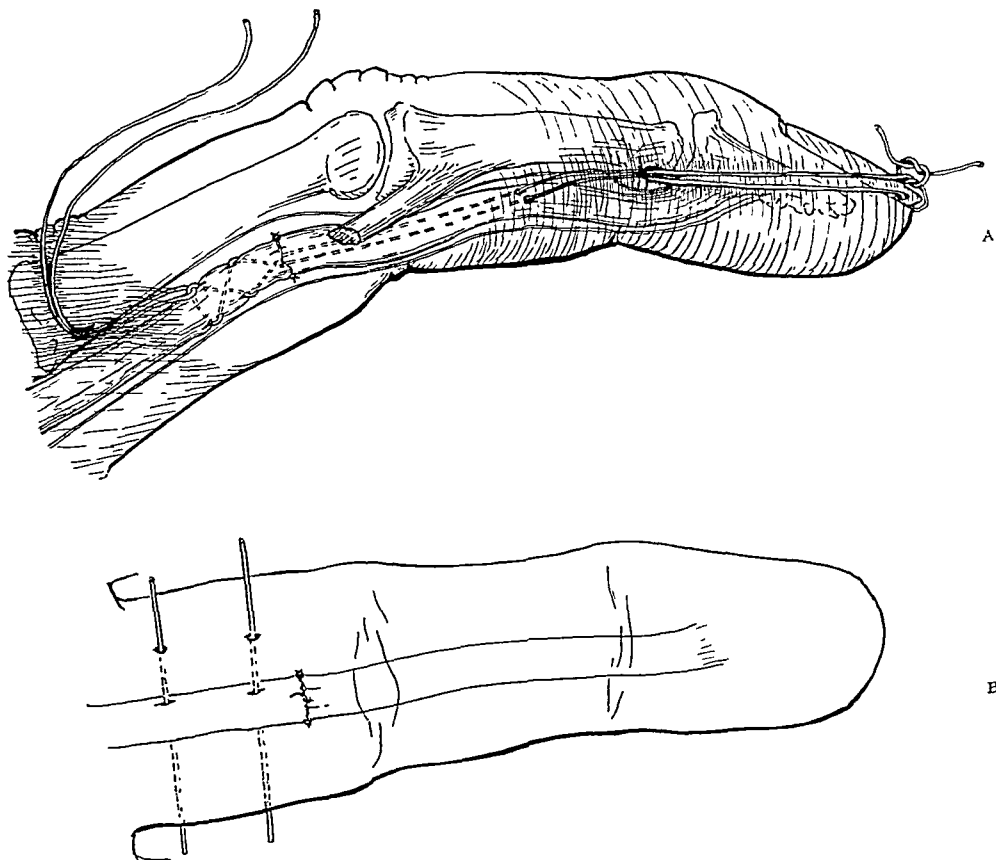
FIG. 3. A, primary suture of flexor profundus in proximal segment of finger. Use of stainless steel wire as a removable suture. Suture wire holds proximal tendon end distalward by passing through the distal tendon, then down the tendon sheath and out through the skin, being attached to the finger nail. The pull out wire is left in place ready to withdraw the suture wire in three weeks when it is clipped off at the skin. Only the profundus tendon is sutured. The sublimis is withdrawn. The annular ligament is slit laterally to allow circulation of the tendon ends during the stage of swelling. This stitch allows for some postoperative exercise. B, simpler method of joining a tendon, holding the proximal tendon end distalward by two or three steel pins. Tendon ends are approximated by a few tiny sutures of stainless steel wire No. 36 or finest silk size .0025. Method does not allow early movement. Tendon may later be freed.

double-pointed scissors excise first the skin edges and then systematically the whole surface of the wound 1 or 2 mm. deep or more if the tissue is too badly traumatized. .

Chisel off infected bone ends thinly. Shave off infected ends of tendons and nerves, removing all the iodine stained tissue, so that there is throughout a surgically aseptic wound of viable tissue.

3. *Covering over Vulnerable Parts.* Tendons, nerves, joints and bones cannot live exposed to the outside world, and when infected cause sloughing, months of dressings and final crippling. In the
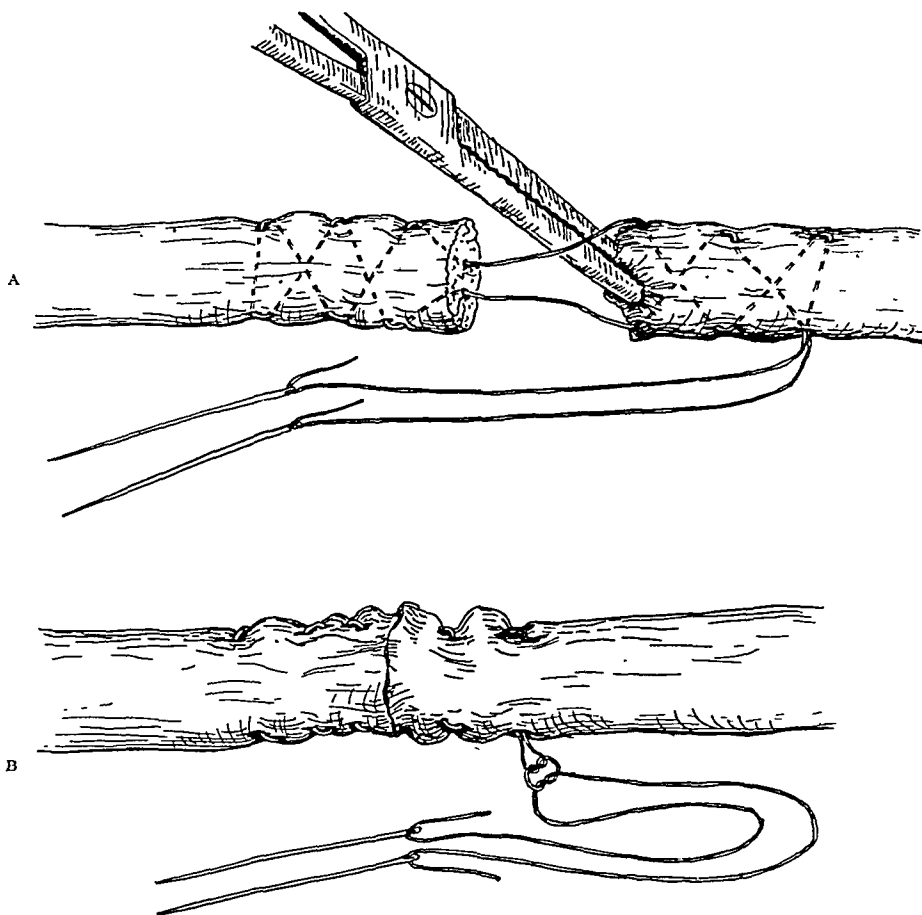

FIG. 4. Method of suturing tendon with stainless steel wire No. 34, leaving the suture permanently in place. A, placing the stitch. The tip grasped by hemostat is clipped off. Care must be used to avoid any kink. B, the tendon ends are bunched well together by sliding them over the smooth wires which are pulled alternately. This prevents later separation of the tendon ends. The wires are knotted together, allowing the knot to sink into the tendon.

golden time before germs have multiplied, all these structures should be spared by the method of covering them over by plastic swinging of skin flaps from the immediate neighborhood. The denuded areas from which the skin flaps are swung should be covered by immediate Thiersch skin graft. By this simple maneuver the saving from an economic standpoint is tremendous.

The tourniquet is then removed and hemorrhage checked by steady pressure for a few minutes and a minimum of fine ties. The

skin and dead spaces are closed, preferably by stainless steel wire No. 35. Buried stitches are omitted or reduced to a minimum. Drainage by slender rubber tubes is used only if seepage is from a large
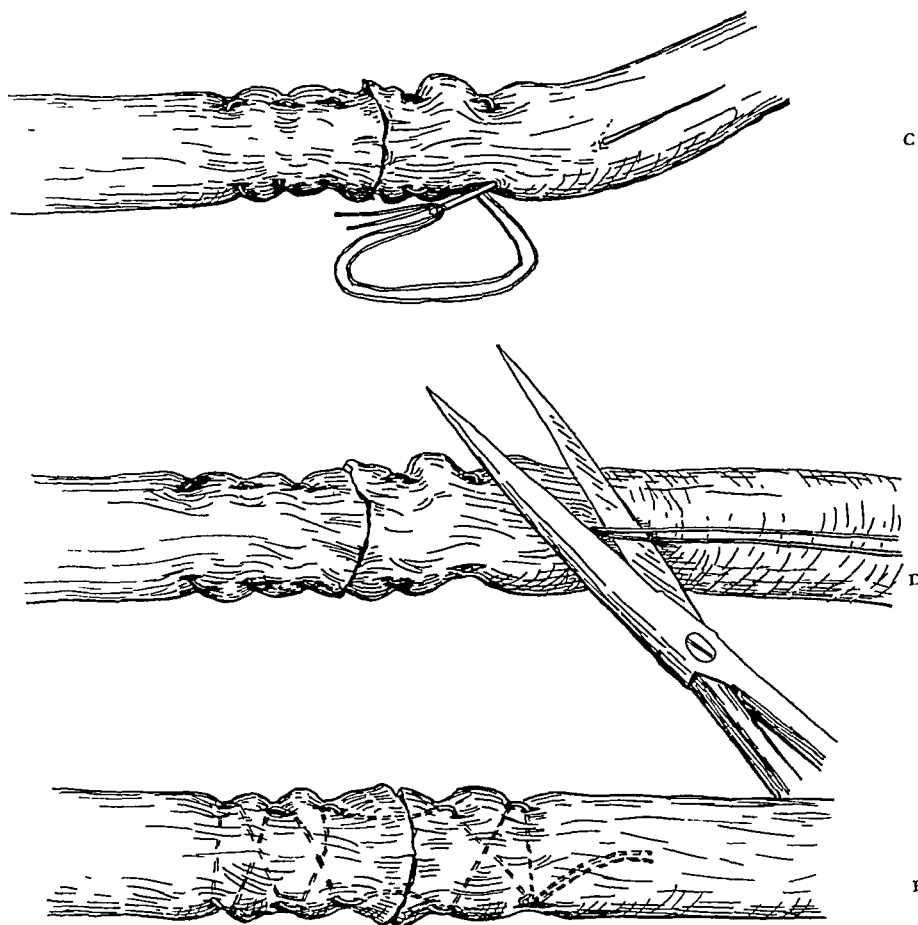

FIG. 4. C, D and E, method of burying the loose ends of the wire in the center of the tendon so that they will not scratch the sheath. This suture with No. 34 wire has tensile strength of 5 pounds which is enough for early protected movement.

area and even then, drains are removed the following day. The limb is immobilized in plaster of Paris. Mild gauze pressure is applied for a few hours or until next day to prevent hematoma. The wound is then allowed to dry uncovered, protected only by a wire cage and gauze or towel covering over that. Even dry gauze dressings become moist in twenty-four hours and encourage infection to enter along the stitches. Dryness prevents this. An injection of 1500 units of antitetanic serum should be given.

Primary suture of tendons should not be done except under these strict indications of time factor, condition of wound, hospital

facilities and special training of surgeon. If these are not available, the wound should be debrided and closed and vulnerable parts should be covered over. No attempt should be made to unite

Fig. 5. Result of primary suture of flexor tendon in proximal segment of a finger with a stainless steel wire removable suture as shown in Figure 3A.

tendons. They can better be repaired later, a month following healing per primam, or if infection has occurred not less than four months after all wounds have healed. A delay of six months is necessary if there has been infection of bone. Infected sutured tendons result in terrible infection that extends rampant up tendon sheaths and spreads through a limb, resulting in crippling or even death. A tendon should never be sutured after twenty-four hours and never in wounds that are traumatized or dirt ground and never without thorough debridement. One crumb of necrotic or infected tissue may cause infection. Skin should never be closed if soiled or devitalized tissue is in the depth or serious infection will follow. Old wounds excised after twenty-four hours should not be sutured closed. A limb should be immobilized continuously and without interruption until the danger of infection is over and should be kept

elevated to prevent edema and poor circulation. One should not seek primary closure if tissues are mangled or dirt ground.

NORMAL TENDON FORMATION

A tendon is enclosed in a thin, close, tight-fitting membrane called epitenon. Interlacing inwards from this between the bundles of tendon cells are connective tissue septa called endotenon. Where the tendon runs straight it is, instead of being enclosed in a tendon sheath, surrounded by paratenon tissue. This is a loose specialized sort of fat lightly attached to the tendon and also to the surrounding fascial tunnel. It is loose and gliding to allow ample excursion of the tendon through it. Where, however, a tendon rounds a corner, as opposite a joint, or around a bony prominence, a tendon sheath formation is present. Here the tendon is free, hung only by a loose sheet of blood vessel bearing mesotenon, or, as in the fingers, by narrow vinculae. The sheath at each end forms telescoping folds, called plicae, which allow for the to and fro motion of the tendon. In sheath formation when a tendon is severed the ends retract far. Their ends merely round over and are found unattached and free in the sheath. Severing in paratenon results in less retraction but rapid proliferation of the connective tissue in and about the tendon end so that a long flame-like projection reaches out and attaches to whatever tissues are there or to the opposite tendon end, if possible, in nature's effort to repair the damage.

HEALING AFTER SUTURE

The healing of tendons has frequently been described, this has been especially well done by Mason and Shearon. Soon after the tendon ends are sutured there is great proliferation of the connective tissue elements in and around the tendon—endo, epi and para—resulting in the rapid formation of a gelatinous fibroblastic splint between tendon ends well established in four days and quite advanced in two weeks, but without strength. Tendon cells are seen to proliferate in four days and in two weeks have bridged the gap. They furnish the strength so that in three weeks splints can be removed and in four weeks the danger of breaking is over. There is much swelling in the first two weeks. Thereafter, the connective tissue elements undergo a loosening process around the tendon to allow movement. Healing in paratenon formation is more active than within a sheath because of the surrounding vascularity.

Problem in Suturing a Tendon in a Sheath. When a tendon severed in sheath formation is primarily sutured, theoretically there

should be certain advantages, namely, the muscle can easily be drawn out, a synovial lined sheath will still be present and the gap between the tendon ends will not be filled with scar tissue as in late repair. It will be found, however, that the tendon juncture will grow fast to the sheath as there is rapid exuberant growth of the connective tissue elements in an effort to establish attachment to something.

A great indirect cause of attachment is that the tendon ends, where sutured together first, go through a stage of swelling. This is caused by the natural healing, the double trauma of the laceration and the surgery, and also the reaction produced by the suture material itself. Consequent ischemia from swelling in an unyielding sheath results in necrosis which is of necessity followed by cicatricial adhesion. A common example of this from excessive trauma is seen in the usual binding of a tendon by adhesions opposite a fractured phalanx.

A tendon will, however, tolerate a degree of surgery and still retain its function. Thus we can open a tendon sheath along the side of a finger, thoroughly exposing and even handling the tendon. The tendon sheath and finger can then be closed and good function will result. It is evident, therefore, that to solve the problem of repair in a tendon in its sheath we must develop a method of minimal trauma to lessen tissue reaction. The narrow caliber of the firm tunnel of the tendon sheath at the place of suture is a factor in causing adhesions. Therefore, such a fascial tunnel should be slit laterally to allow the tunnel to expand to a large enough caliber to accommodate for the necessary swelling of tendon repair.

A primary tendon repair provokes so much cicatrix that all structures should be handled with minimal trauma. The ends are joined with the simplest stitch and with suture material which is least irritating. In late tendon repairs the stitch may be more elaborately spliced into a length of tendon so it will withstand the strain of early exercise, but in primary repairs the suture should be the simplest that will hold the tendon ends lightly together in good approximation. To prevent breaking we should rely more on splinting than on suture material.

The material of suture is important. Chromic catgut is prone to infection, specific sensitization and cicatricial reaction. O'Shea, in reviewing 870 tendon sutures, reported infection with it six times as frequently as with silk. Strong braided fine untreated silk No. 2 has been the usual suture of choice. However, this material is a foreign body which creates tissue reaction resulting in adhesions which

are our main cause of failure in suturing a tendon in a sheath. We should therefore select a suture material that gives the least tissue reaction.

From the study of Venable and Stook, vitallium and stainless steel were found to be the least irritating to tissues as they are non-electrolytic. There is available excellent very thin flexible wire of stainless steel. For three years I have used this continually for skin suture and am enthusiastic over its freedom from irritation in the skin where we can clearly see it. We often cover it with a plaster cast for months without concern, for it does not cause irritation. Babcock has popularized its use in infected wounds and has shown that granulation tissue will grow over and bury it. Of all suture materials stainless steel causes the least reaction and it is therefore logical to use it for tendon suture where adhesion-forming irritation is especially to be avoided. If placed centrally in a tendon with the free ends buried in the center of the tendon so that they will not scratch, the wire may be left in with confidence of better tissue toleration than in the case of silk. For the mechanical effects more time is needed.

A simple stitch, with a needle at each end of the wire No. 34, first penetrates the tendon transversely. The needles, after crossing around a few tendon fibers, then pass through the tendon diagonally from opposite sides and again penetrate diagonally to emerge from the center of the tendon ends. The two wire ends are then pulled to remove all slack, the wire sinking out of sight into the tendon. With the tip of the opposite tendon end held taut in forceps (and later clipped off) the two wires are similarly placed, starting at the tip and emerging from the same point farther down the tendon, having passed diagonally through twice and one of them also transversely. The second tendon end is then slid down these smooth wires and tied to the other, the knot burying itself in the tendon remote from the suture line. The two wires are then threaded through the depth of the tendon and cut off. Such a stitch withstands 5 pounds. Care should be used to avoid kinks as these cause the wire to break. The tendon ends have been bunched together so they will not separate. Approximation can be made exact if necessary by a tiny stitch of .0025 silk or No. 36 wire.

There is never trouble making tendon ends unite, but often trouble from outside adhesions. Therefore, it is better to have the stitches buried than irritating at the sides of the tendon where we do not want adhesions. A central core suture is not objectionable as all necrosis from the grasp of the stitch is eventually displaced

by tendon tissue. This necrosis is better central than peripheral, since the latter would cause adhesions. Nowhere is the full thickness of the tendon strangled of blood supply and the ends of the tendons themselves are free from suture for good repair.

We have also been using the wire suture removable from the outside. This is not difficult considering that it is only the proximal end of the tendon that pulls, the distal tendon end being merely passive. The stitch is placed in the proximal end only and the two wires emerging from the tendon end are passed on up through the center of the opposite or distal tendon end and out through the skin to a firm attachment in the finger nail or to an adhesive plaster applied broadly to the skin. Tiny sutures of No. 36 hairlike wire accurately approximate the tendon ends, but the main wire stitch holds the proximal tendon end distalwards so the tendon ends cannot pull apart.

In order to remove the wire three weeks later, a second wire is threaded through the proximal loop of the suture wire and its two ends threaded on a straight needle are passed proximalwards up the tendon sheath a short distance and then out through the skin and left there. This is the pull out wire. When in three weeks the ends of the suture wire are cut off close under the skin where they emerge, the pull out wire easily withdraws the suture wire right out of the tendon and to the outside as it pulls it by its proximal loop. If there is resistance, a light rubber band left on will withdraw the stitch without danger of breaking. This arrangement allows for moderate excursion of the tendon in its sheath during healing in order to prevent adhesions to the sheath.

Postoperatively, adhesions are guarded against by exercise. Flexing the joints distal to the tendon suture pushes the tendon proximalward in the sheath. Thus the flexor tendon is pushed an inch into the palm by passively flexing the finger. As both the suture wire and pull out wire have been passed for some distance through the sheath before emerging through the skin, the wires will not hinder this motion nor the natural pulling motion of the tendon. Such a stitch is useful in the hands and feet and in joining tendons to their insertion, especially in suturing within a sheath.

A simpler method is to transfix the proximal portion of the severed tendon through and through the finger or limb with two or three stainless steel pins. Two pins in a finger will prevent the proximal tendon end, up to 5 pounds, from pulling away from the distal end. A tiny approximation stitch of No. 36 wire should also

be used. This method results in some adhesions to the sheath as it does not allow motion. Later the adhesions may be freed.

With the above methods reliance should not be placed on the strength of suture, but instead on immobilizing the joints in flexion by plaster of Paris to leave the involved muscle in complete relaxation. In the case of a flexor tendon in the hand or wrist, only the wrist should be flexed in this way and not the fingers. The muscle will then not be able to move forcefully enough on the tendon to separate the sutured ends. However, there is enough weak motion to stimulate growth and lessen adhesions while physiologic union is occurring.

Extensor tendons in hand and forearm are successfully united by the simplest figure-of-eight stitch of stainless steel wire, passing through the skin as a mattress suture and forming the deeper loop of the figure-of-eight through the tendon ends to hold them together. Both wrist and digits are then held in almost full dorsiflexion for three weeks, when the stitch is removed; another week of less dorsiflexion is then allowed.

If only the flexor sublimis of a finger is severed, it is best to let it alone. Suturing it will cause it to adhere to the profundus. If both sublimis and profundus tendons are severed, the sublimis should be allowed to retract and the profoundus alone be sutured. If both are sutured they will become fast to each other and so will act on only the middle but not the distal joint of the finger.

CONCLUSIONS

Primary repair of tendons should not be done except under strict indications covering the nature of the wound and adequate facilities of both hospital and surgeon. Operation must be prompt, debridement thorough and all vulnerable parts should be closed over. Repairing within a sheath is the most difficult. Trauma should be minimal and the tendon stitch simple and of non-irritating material, preferably stainless steel wire. This may be left in the tendon or be placed in such a way that the wire is removable.

DISCUSSION

HOMER D. DUDLEY (Seattle): It has been my privilege on a number of occasions to visit Dr. Bunnell and see his work. I have always been fascinated by everything he does, and have about concluded that he is as much a magician as a surgeon. I no sooner learn one of his intricate methods than he springs a new one on me. The last time I saw him he told me about

his use of silver wire. I have had occasion to use wire three times since then. The results are yet to be seen.

The problem of suturing severed tendons of the hand, especially that portion in rigid tunnels, has not been solved satisfactorily. Many forms of treatment and definite technique have been recommended to meet this condition. There is no difference of opinion as to the exact fundamental requirements for success in suturing severed tendons. Obviously, the type of suture material and its application surgically does not solve the problem as a whole. Under ideal conditions, utilizing all the fundamental principles, there is still an element of failure. The end result, therefore, is modified by one or another of those factors over which the surgeon frequently has no control. The type and location of the injury, element of time, age of the patient, conditions incident to primary and surgical trauma, infection, preparation of the wound, together with the type of suture material used and technique employed are only some of the factors. There still remain those anatomic and physiologic conditions peculiar to tendons that predispose to formation of adhesions.

A study of the physiology and anatomy of tendons is helpful in understanding the necessity for atraumatic technique, also a knowledge of tendon tension and its application in primary suture and postoperative care.

Because of the tendency of severed tendon fibers to rapidly proliferate and attach themselves to some neighboring structure, a most exacting technique is demanded. Various measures and agents to prevent the formation of adhesions have been employed. These may be divided into two groups: that method whereby some tissue has been employed to surround the site of tendon injury such as fat, fascia, blood vessels, various forms of animal membrane, and so forth; and the second, in which some non-resorbable foreign material has been used to reconstruct the tendon sheath such as celloidin tubes, modeled cartilage, magnesium, and stainless steel rods. All of these have offered some improvement. The former are practical chiefly in the primary repair of tendons, not in fibrous spaces, and the latter as secondary or reconstructive measures.

All are agreed that regardless of these measures, certain fundamental principles are essential for a minimum of success, as atraumatic technique, avoidance of infection, complete hemostasis, accurate apposition of severed tendon ends, the use of non-irritating antiseptics and suture material, appropriate dressings, splinting and postoperative care. When one realizes the importance of employing all the fundamental requirements for success in primary repair of severed tendons, it is difficult to evaluate any one measure as being more important than another. Suture material is only one of these and best of all is the one least irritating. Here stainless steel wire may prove most valuable.

Dr. Bunnell has offered an ingenious method which may improve one or more of these requirements and likely to better functional end results. I would like to ask Dr. Bunnell if he would consider it applicable to use

removable wire sutures to hold severed tendon ends in their normal relationship, when the time for ideal primary repair had passed and in the period when infection is likely to occur. One might fix the proximal end of the tendon and bring the wire out at a distant point, fix it in place and close the skin. If the wound did not become infected, the wire would hold until the appropriate time for secondary repair. If infection occurred and it seemed desirable, the wire suture could be removed easily.

LAURIE H. McKIM (Montreal): As Dr. Dudley has just stated, we no sooner get accustomed to a very careful technique of Dr. Bunnell's than he brings out something that sounds even better. We all know the work he has done, and I think the majority of us believe that whatever suture material Dr. Bunnell happens to use, he will probably have a little better result with it than we will. I am sure all who have taken notice of his previous work, his direction to the great need for careful technique and the avoidance of trauma, believe that this new departure, the use of steel wire as a suture material for tendons, may prove valuable.

The introduction of steel wire in tendon work brings up the whole question of the pathology of foreign body reaction. We may have to revise some of the opinions we have held. We know that foreign body reaction is probably of three kinds—chemical, allergic, and electrochemical. There are probably others, but we have accustomed ourselves to grouping all together as simple "foreign body reaction." It would seem that Dr. Bunnell's stainless steel wire has possibly overcome the majority of the factors concerned in this reaction.

My own practice in the past ten years has been, unless the case is seen within two or three hours, not to suture any tendons on the dorsum of the hand. I place the hand in a plaster of Paris "cock-up" splint to the ends of the fingers. I leave the cast on for six weeks. I have yet to get a failure of union, and I have yet to get other than absolutely perfect function in any case.

I should like to ask Dr. Bunnell whether he considers that the use of stainless steel wire would be equally applicable in the insertion of tendon grafts.

STERLING BUNNELL (closing): I wish to thank the discussers.

The problem of primarily suturing a tendon in a sheath is not yet solved. I do not claim a solution, but am striving toward some method that will improve our results because they are unsatisfactory the world over. I hope by this paper merely to direct the trend of thought along a line that will give better results.

The use of stainless steel wire is, of course, not the only factor. It is just one and there are many others which are essential for success.

I think Dr. Dudley's suggestion is excellent in that in some instances where elaborate suturing is inadvisable one can merely place a figure-of-eight stitch or do something simple, temporarily to hold the two tendon ends together until secondary repair is done, just as we often do in primary

nerve repair. This will keep the muscle from contracting. Usually after two months tendon ends can be drawn together but not after three months. An objection is that two operations leave more adhesions than one.

It is very important in placing these wire stitches never to kink the wire, and that is up to an assistant to hold the slack of the suture, because any kink that forms in the wire will make it break.

Dr. McKim mentioned tendon grafts. The wire acts in free grafts just as well as in any tendon. There is no essential difference.

TRAUMATIC LESIONS OF THE NERVES OF THE WRIST AND HAND

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A MAIMED hand is a calamity. If it befalls an individual of financial independence, he is subjected to the embarrassment of the unsightliness of the member and is deprived of
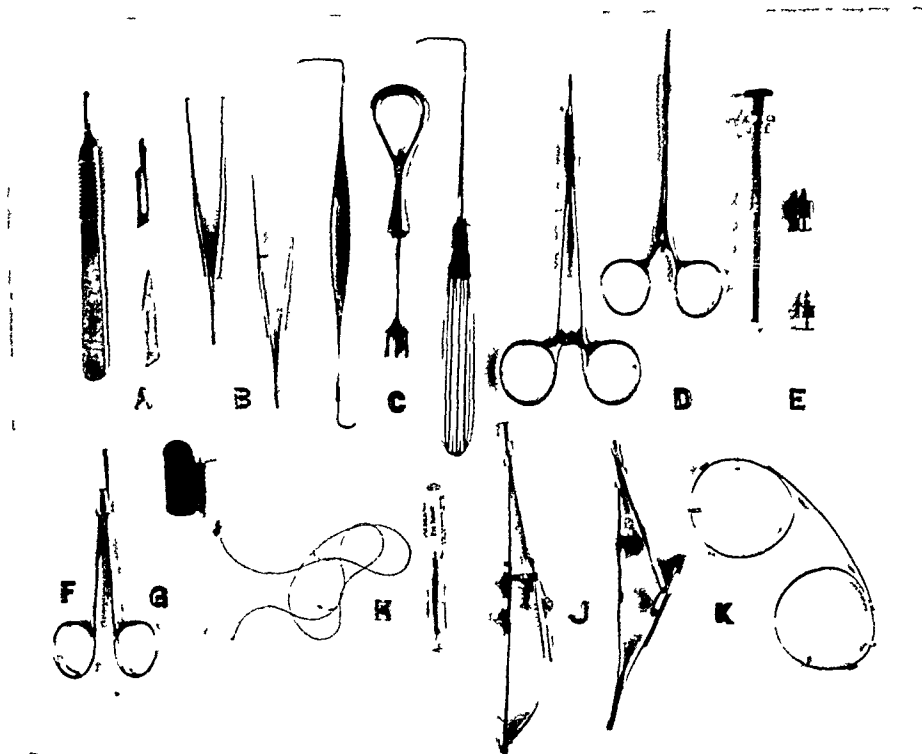
FIG. 1. Armamentarium. Fine instruments used to supplement the usual dissecting kit. A, knife handle and blades B, fine toothed forceps. C, retractors. D, hemostats. E, syringe and needles for nerve injection. F, scissors. G, dropper for neurorrhaphy. H, fine needles carrying 4-0 and 6-0 silk. J, needle holders. K, magnifying lenses which may be clasped over spectacles.

participation in certain activities which contribute to his happiness. If it befalls an individual who must rely entirely upon his hands for his livelihood, the physical affliction may so seriously alter his economic status that he and his dependents may be reduced to a
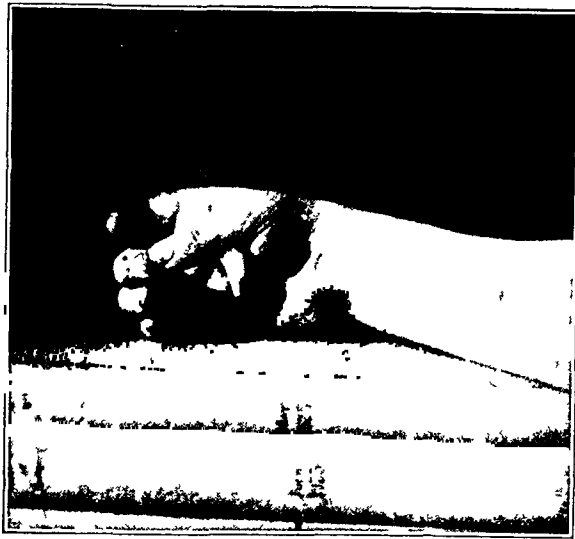
FIG. 2A.

FIG. 2B.

FIG. 2. Median neurorrhaphy and tenorrhaphy at wrist. Girl, age 3, received severe wound with glass across anterior surface of wrist, dividing all structures except the ulnar nerve and flexor carpi ulnaris tendon. Repair several hours after injury. Photographs (A, B and C) taken eleven months later show perfect apposition and flexion and extension of fingers. The case is interesting on account of the small size of the parts and because of the use of squeaking rubber toys to secure active movements. (From Harmer, in *Boston M. & S. J.*, 194: 739, 1926.)

wretched existence. Disabilities may be due to several causes. It is my purpose to consider those disabilities consequent to trauma of the nerves of the wrist and hand. The prevention of disability
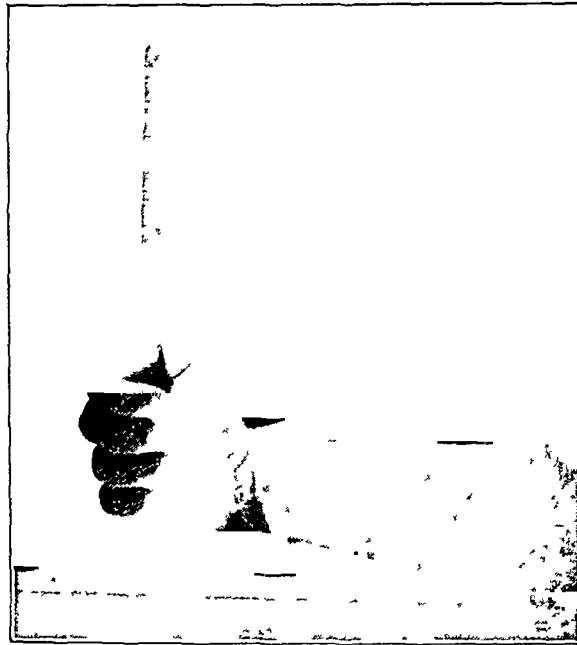
Fig. 2c. For descriptive legend see opposite page.

depends upon the immediate recognition of the nature and extent of the injury and upon immediate repair. When the disability is established, amelioration depends upon the recognition of the underlying pathology and upon reconstruction undertaken sufficiently early to insure a reasonable expectancy of nerve regeneration.

Anatomy. The nerves which may be injured are the median nerve with its branches, the ulnar nerve with its branches, and the distal branches of the radial cutaneous nerve. The *median nerve* at the wrist lies between the palmaris longus (when present) and the flexor carpi radialis but at a somewhat deeper level. It passes beneath the transverse carpal ligament (volar retinaculum) and approximately at the distal margin of the ligament breaks up into branches like the crown of a tree. The lateral group consists of three branches. The first is the so-called motor nerve to the thumb, which crosses the radial bursa and supplies the superficial muscles of the thenar eminence (abductor pollicis, apponens pollicis and the superficial head of the flexor brevis pollicis). The other branches of this group are distributed to the two sides of the thumb and the radial side of the index finger. From the latter nerve arises the twig to the first

lumbrical muscle. The medial division consists of two branches which run distally towards the clefts between the index and middle and the middle and ring fingers. They separate into digital nerves for

FIG. 3. Neurorrhaphy (median and ulnar) and tenorrhaphy (flexor carpi ulnaris, all of profundus, two of sublimis and palmaris longus) performed twenty-four hours after injury (thrusting arm through pane of glass). Pictures taken five months after operation show extent of wound, development of thenar and hypothenar eminences, action of intrinsic muscles supplied by both nerves, and flexion and extension of fingers. Note rapidity of regeneration in this high school athlete. (From Harmer, in *S. Clin. North America*, 1: 809, 1921.)

the adjacent sides of these digits. From one is given off a twig to the second lumbrical. Although these palmar digital branches of the median nerve pass deep to the superficial volar arch, they ultimately come to lie to the volar side of the digital arteries in the fingers. It is to be remembered that these nerves also supply the distal portions of the dorsal aspect of the thumb to the base of the nail, of the index finger to the distal joint, and of the middle and ring fingers to the middle joints.

The *ulnar nerve* at the wrist courses between the tendon of the flexor carpi ulnaris medially and the ulnar vessels laterally. It then passes in a special compartment superficial to the carpal ligament, skirting the radial margin of the pisiform. Towards the distal margin of the ligament it divides into its superficial and deep branches. The superficial branch passes beneath the palmaris brevis muscle, which it supplies, and then splits into a medial division which innervates the medial margin of the hand and little finger, and a lateral

division which, coursing on the hypothenar muscles, splits to supply the contiguous margins of the ring and little fingers. The deep branch of the ulnar nerve with the deep branch of the artery sinks into the interval between the abductor and flexor brevis minimi digiti muscles, supplying these and the apponens minimi digiti. It then passes laterally on "bed-rock" beneath the flexor tendons of the fingers paralleling the deep palmar arch. By this route it reaches the deep muscles of the thenar eminence (the adductor obliquus and transversus pollicis and the deep head of the flexor brevis pollicis). On the way across the root of the palm it sends two branches to each interosseous space. These branches innervate the three palmar and four dorsal interossei and the two medial lumbricals. It is to be remembered that there exists an anastomosis between the superficial branches of the ulnar nerve and the median nerve at the root of the palm and another between the deep branch of the ulnar nerve and the median nerve at the base of the thumb. The frequent double innervation of the third lumbrical muscle should also be borne in mind.

There remain to be described the cutaneous nerves of the palm and of the dorsal aspects of the hand and digits. The palmar cutaneous branches of the ulnar and median nerves arise at or above the wrist, pass superficial to the transverse carpal ligament and are distributed to areas of the palm, generally speaking, medial and lateral to the long axis of the fourth metacarpal. The skin over the thenar eminence is, however, supplied by a distal continuation of the lateral cutaneous nerve of the forearm and by branches from the superficial ramus of the radial nerve.

The large sensory branch of the radial nerve gains the dorsal aspect of the lower forearm by passing beneath the tendon of the brachioradialis. Its four branches are distributed as follows: the radial margin of the hand and thumb, the medial margin of the thumb, the lateral marg n of the index finger, and the adjacent sides of the index and middle fingers. It should be remembered that there is another twig which joins a branch of the dorsal cutaneous nerve of the ulnar to supply the contiguous margins of the middle and ring fingers. It should also be remembered that these digital branches extend on the thumb to the base of the nail, on the index finger to the distal joint, and on the middle and ring fingers only to the middle joints. The posterior aspect of the hand and the medial digits are supplied through the dorsal cutaneous branch of the ulnar nerve. This nerve arises from the parent nerve about 2 inches proximal to the ulnar styloid. It then winds about the medial margin
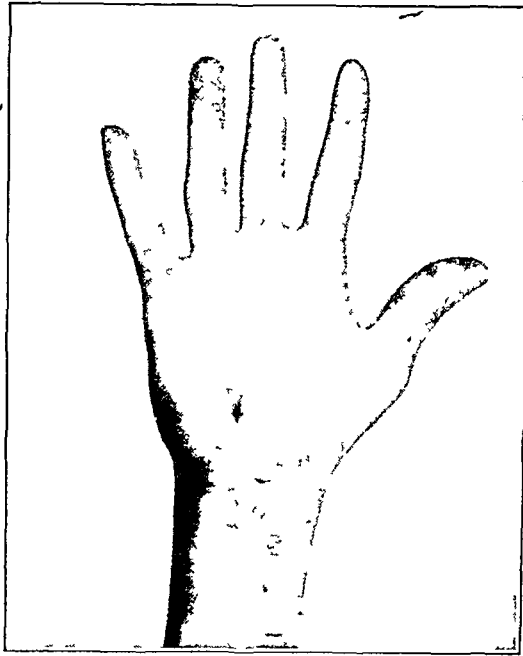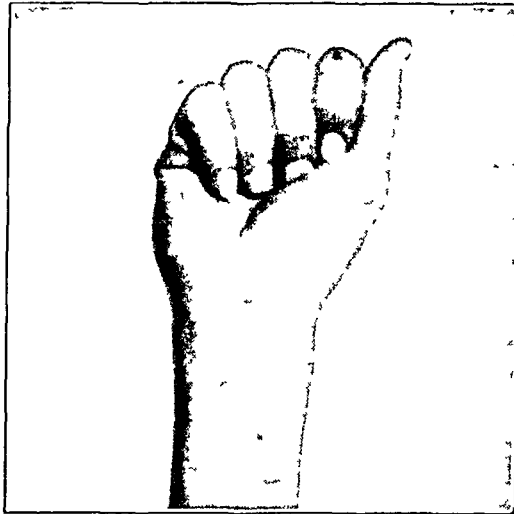
FIG. 4A.

FIG. 4B.

FIG. 4. Neurorrhaphy (median and ulnar) and tenorrhaphy (all flexors) at wrist performed several hours after injury (thrusting arm through a pane of glass). Pictures taken nineteen months after operation show development of thenar and hypothenar eminences, action of intrinsic muscles supplied by both nerves, and flexion and extension of fingers. (From Harmer, in *Boston M. & S. J.*, 194· 739, 1926.)

of the hand a thumb's breadth distal to this process and divides into three branches. One of these innervates the medial margin of the hand and little finger, another continues on the dorsum to supply

FIG. 4C. For descriptive legend see opposite page.

the contiguous margins of the little and ring fingers, and the third continues distally to anastomose with a branch of the radial to supply the contiguous margins of the ring and middle fingers (as noted above).

Preoperative Examination. A careful examination should be made in all cases to gain an intelligent understanding of the damage before anesthesia is started. Since a nerve injury is rarely an isolated lesion but is usually accompanied by damage to other structures, the examination must be of the entire hand and wrist. With practice and gentleness this can be accomplished quickly and with very little discomfort to the patient. The repair of a lacerated wound, even an extensive laceration on the volar aspect of the wrist, should not be an exploratory operation.[3]

The situation and extent of the wound are noted and it is then covered with sterile gauze. The functions of the parts are tested. The surgeon executes with his own hand the movements which he wishes the patient to attempt. Beginning with the thumb, flexion and extension of each phalanx of each digit are tested. To this end it is helpful to support the joint proximal to the phalanx which is being tested. Except for the extension of the middle and terminal phalanges of the fingers, these are gross movements. The muscle bellies of these tendons are innervated in the forearm. To determine the

integrity of the ulnar and median nerves at the wrist, the intrinsic muscles of the hand must be tested. In case of the ulnar nerve these tests concern the ability to adduct and abduct the fingers and to

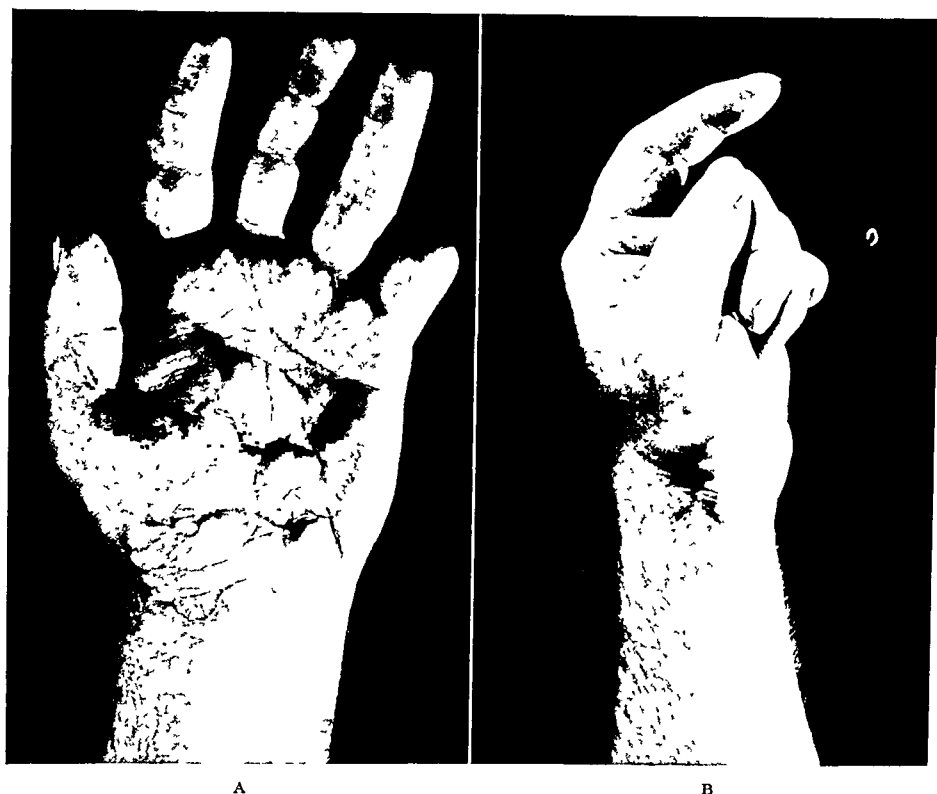A   B

FIG. 5. A and B, neurorrhaphy (median at base of palm) and tenorrhaphy (flexor carpi radialis, flexor sublimis and profundus of index finger, and palmaris longus) in palm and at wrist, performed two hours after injury (from gouges of a wood-fluting machine revolving 6000 times a minute). The soft parts were widely extruded through several more or less parallel undulating deep wounds, including lacerated muscles of the thenar eminence. Pictures taken five and one-half months after operation. Show extent of lacerated wounds, active apposition of the thumb and range of active motion of middle and terminal phalanges of index finger.

adduct the thumb, the ability to flex the fingers as rods at the meta-carpophalangeal joints, and the ability to extend the middle and distal phalanges of the middle, ring, and little fingers, especially the ring and little. In case of the median nerve the functional tests concern the ability to appose the thumb to the tips of the fingers, to abduct (not extend) the thumb, and to extend the middle and ter-minal phalanges of the middle and index fingers, especially the index. Although tests for cutaneous sensation may be of value in estimating the repair problem in lesser injuries and in reconstruc-tion work on the hand, the writer believes that the mental attitude

of the patient often renders them unreliable in extensive acute injuries. It is true that patients will at times volunteer the information that certain parts feel "numb" or "dead" and operative find-

FIG. 5c. Superimposed photograph, showing action of first lumbrical through median nerve. Note: full flexion of the terminal and middle phalanges of the index finger is prevented by scar tissue at the root of the palm and wrist. Will probably require correction by subsequent operation. Amputation of little finger from previous accident.

ings will substantiate the accuracy of their observations. In cases of injury to the rami of the radial cutaneous or of the ulnar dorsal cutaneous nerves, sensory tests, of course, are indispensable.

The necessity for thorough preoperative examination is well illustrated in *multiple wounds*.[4] Wounds which seem trivial may be serious. Apparently insignificant wounds of the wrist may sever the median or ulnar nerves without injury to contiguous tendons. Unless special tests for these nerves have been made, their injury will be overlooked when operation for other wounds is undertaken. Subsequent neurorrhaphy will be necessary. It is needless to state that immediate repair of a nerve is not only much less difficult but also more likely to succeed.

The integrity of circulation may be determined by noting the radial and ulnar pulses at the wrist, the color and warmth of the hand, and the capillary blush in the nail-beds.

The appearance of the hand following *old injuries* of the median and ulnar nerves at the wrist is so characteristic that the diagnosis is at once apparent. In ulnar palsy the ring and little fingers are hyperextended at their metacarpophalangeal joints and flexed at their middle and distal joints. The hypothenar, deep thenar, and
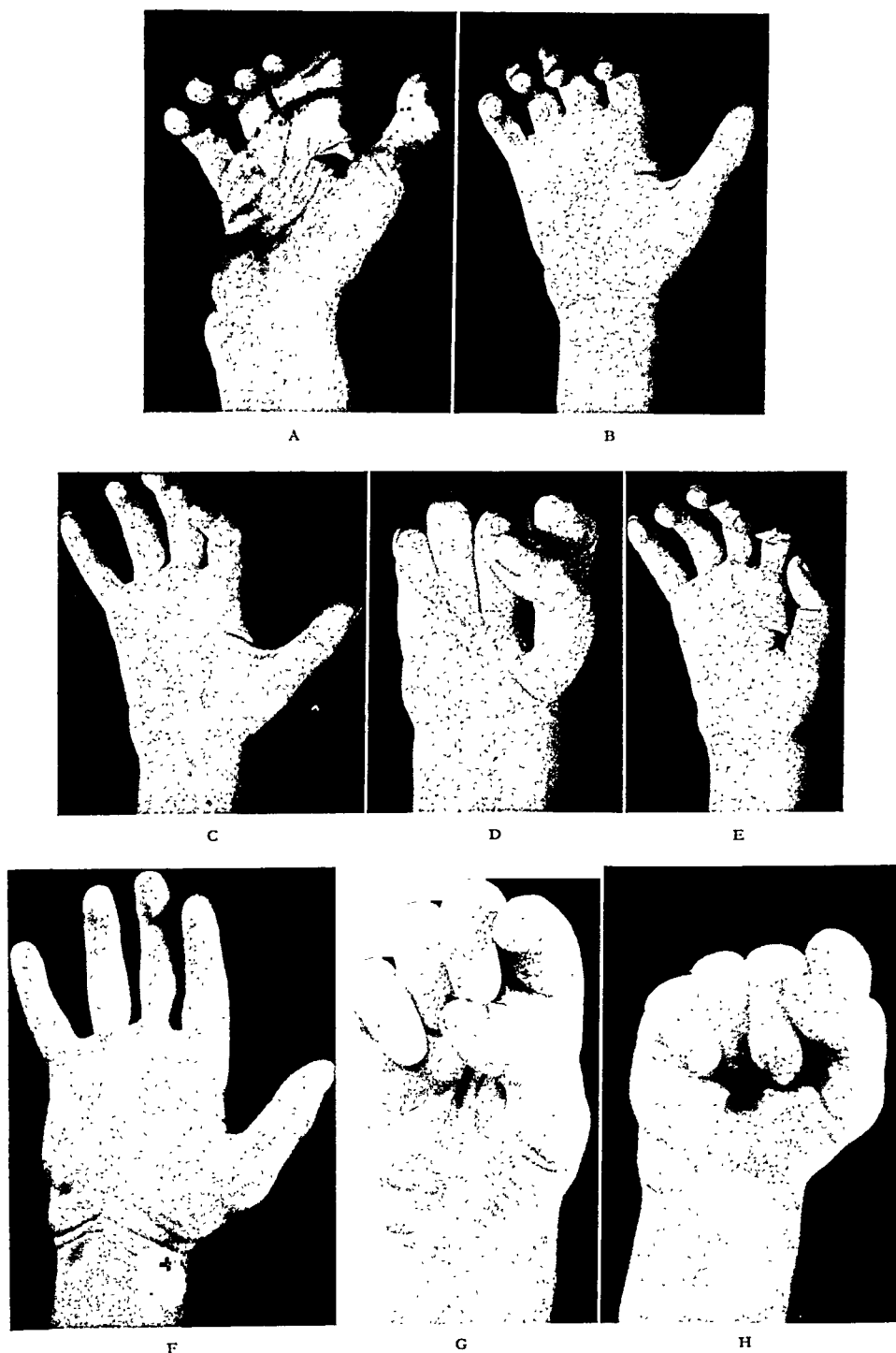
FIG. 6. Late median and ulnar neurorrhaphy and tenorrhaphy at wrist. An immediate repair had been attempted elsewhere eight months previously. A and B, ability to flex and extend fingers and thumb, September 19, 1928. Note atrophy of thenar and hypothenar eminences. Operation, September 21, 1928, showed both ends of median nerve sutured to tendons, ulnar nerve unsutured and other tendon ends matted and bridged by scar. C, D and E, ability to flex and extend fingers and thumb in May 1929. F, G and H, September 10, 1938, showing flexion and extension and ability to appose and adduct thumb. Note devel-

interosseous muscles are conspicuously atrophied. In median palsy the thumb is held in a plane with the palm, producing the so-called simian or ape hand. The superficial thenar muscles are obviously
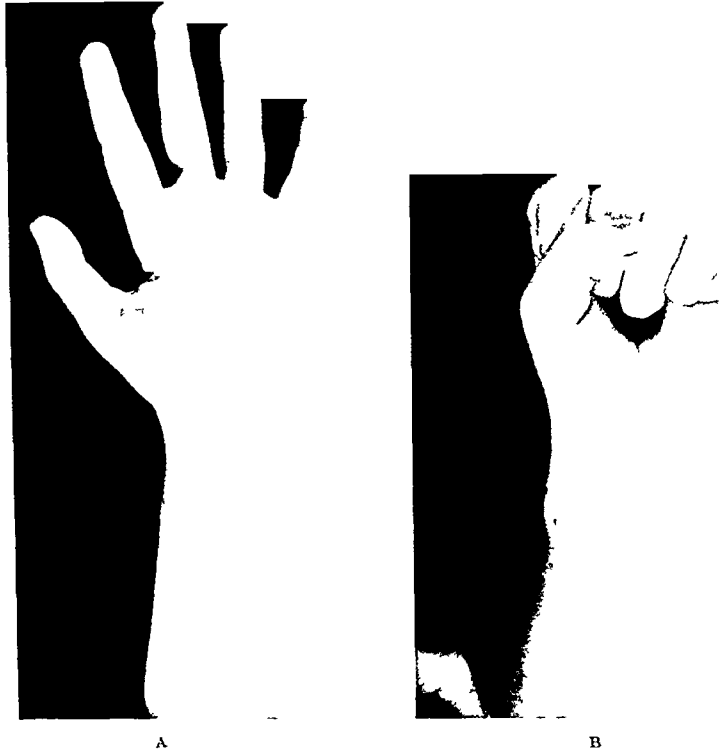

A   B

FIG. 7. Girl, aged 9. Late ulnar neurorrhaphy at wrist. Seen seven
   months after lacerated wound of wrist. Immediate operation and
   another two months later had been done elsewhere. Pictures taken
   nine months after third operation. Show extent of wound necessary
   to effect suture, and action of intrinsic muscles supplied by ulnar
   nerve.

atrophied. When both the ulnar and median nerves have been injured, the hand presents a combination of these characteristic changes and the entire thenar eminence is wasted.

Despite such obvious features an examination of the whole hand, as described under acute injuries, must be made to determine the integrity of other structures. Were any tendons coincidently severed? Have any joints become subsequently impaired? Tests for muscle function must be carefully observed because rebound and supplementary motility may simulate movements which may be expected to be impossible in a particular nerve palsy.[7] For example, relaxation following strained hyperextension at the metacarpophalangeal joints may produce rebound flexion at these joints.

Again, in a median nerve palsy, apposition of the thumb may be simulated by the combined action of the adductor and deep head of the flexor brevis pollicis muscles. Testing regional sensibility by

FIG. 7C. Development of hypothenar
eminence and action of interossei.

pin-prick alone will lead to false deductions regarding the degree of involvement of a sensory nerve. Both epicritic (light touch, slight changes of heat and cold, compass points) and protopathic (pin-prick, extremes of temperature) tests should be employed. It is to be remembered that after the division of a sensory nerve the area of total anesthesia is less than the area of anatomic distribution of the nerve.[5] In the examination of old injuries the presence of neuromata may be revealed by their localized hypersensitiveness. Regional paresthesia may suggest scar constriction of a nerve, for example, the organization of a subfascial hematoma as a sequel to a crush of the hand by rollers.

   Armamentarium. The surgery of nerves is delicate work. Appropriate instruments in good condition are indispensable. The accompanying illustration (Fig. 1) shows a few special instruments which are used to supplement the usual dissecting kit. Most of the work is done by sharp dissection. This causes less trauma than the crushing of scissors. For the same reason no smooth forceps are employed. Curved needles swaged with fine silk facilitate nerve suture and minimize trauma. The magnifying lenses can be clasped

over spectacles and are found serviceable by the author for inspect-
ing the ends of nerves which are being transected in preparation
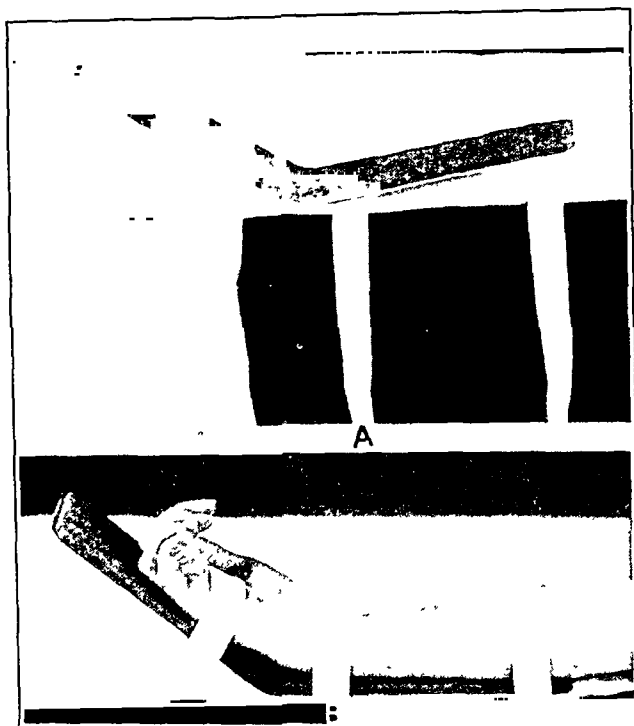for neurorrhaphy following old injury.

FIG. 8. Wrist splint of felt-covered aluminum used follow-
ing repair of nerves and flexor tendons at wrist, A, the
splint has been gently angulated to receive the flexed
wrist. B, splint in use. Although the fingers can be
actively moved, tension on the sutured nerves is pre-
vented. The support of the fifth and fourth metacar-
pophalangeal joints prevents undue stretching of
hypothenar muscles in case of ulnar neurorrhaphy.
The addition of the thumb cot (Fig. 10) similarly pro-
tects the thenar muscles in case of ulnar or median
neurorrhaphy. The splint does not fit the subject, as the
forearm portion is too short and the hand portion too
long. (From Harmer, in *New England J. Med.*, 214:
613, 1936.)

Anesthesia. In most cases general anesthesia is necessary.
The author has had no experience with novocaine block at the wrist
or at proximal levels.

Preoperative Preparation. The preparation for immediate opera-
tion is very important. A hasty or rough preparation may ruin
a pretty operation. In cases in which tendons are coincidently

severed the part is held during this procedure in a position which will prevent their further retraction. In the preparation of bad lacerations or crushes a very thorough but gentle cleansing with

A   B

FIG. 9. Thumb cot of cotton suede to relax the thenar muscles. Used early in conjunction with aluminum wrist splint (Fig. 10) for neurorrhaphy and tenorrhaphy at wrist and later in those cases in which only the median nerve has been sutured (see text). Note double lacing from tip carried between fourth and fifth fingers to back of wrist, looped, carried through hem at base and tied about wrist.

soap and water is followed by prolonged douching with several quarts of sterile salt solution and completed by a douching with ether. During the process frayed tissues which are floated by the douche are snipped off and damaged tissues debrided. Thirty minutes devoted to cleansing of an extensive laceration in this manner are profitably spent. "Patience is a virtue." *Impatience to operate is too common a failing.*[4]

Gloves, instruments, and under-sheet are discarded. The *skin* is painted with half-strength iodine or one of the acetone-alcohol mercurials, and sterile drapes are placed. The hand is elevated several inches on a folded sheet or towels. For reconstruction work the hand and forearm receive twice daily preparations with soap, water, ether, and alcohol followed by a sterile dressing, for one or two days before operation. Shaving is done only at the last preparation.

The indications for antitetanic serum (or gas bacillus serum) and the technique of preliminary intradermal injection for testing sensitivity are the same as for wounds elsewhere in the body.
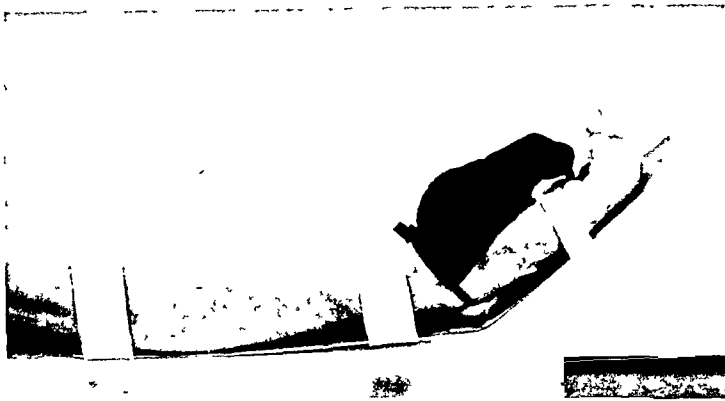

FIG. 10. Combination aluminum wrist-splint and thumb-cot. This combination is used in the early convalescence following neurorrhaphy of the ulnar nerve, median nerve, or both nerves at the wrist or root of the palm. Felt lining of metal splint has been omitted.

Neurorrhaphy in Fresh Wounds. Operative trauma should be minimal. This implies careful preoperative diagnosis, adequate exposure, and gentle and deliberate technique. Attempting to effect a repair through the original traumatic wound almost always necessitates rough handlings of tissues. A generous incision should be made. In dealing with fresh wounds no tourniquet is used. Slight proximal or distal pressure suffices to control bleeding until the incision has been made through the skin and fascia. Vessels are then secured. Practically no further bleeding is encountered.

At the volar aspect of the wrist severance of the median or the ulnar nerve is rarely an isolated lesion. Tendons are usually coincidently divided. (Figs. 2, 3 and 4.) In an extensive wound in this situation both nerves and most of the tendons may be damaged. When the operative approach is completed, the wound margins are retracted. Nothing is disturbed. The structures are studied in situ and identified. The median nerve is recognized by its grayish color, its tiny anterior vessel, its fibered ends and its position. At the wrist it lies just beneath and lateral to the palmaris longus tendon. If the latter is absent, the nerve is very superficial. Distally it lies just beneath the annular ligament. The ulnar nerve is recognized by its fibered ends and its position, at the wrist immediately lateral to the flexor carpi ulnaris tendon and distally anterior to the annular ligament skirting the pisiform. The proximal and distal ends of the

median and ulnar nerves are turned out of the wound and wrapped in small sponges soaked in warm saline solution until the coincidently severed tendons have been sutured. The nerve ends are

FIG. 11A. Flanged cross hand splint of aluminum used in later treatment of ulnar neurorrhaphy or ulnar and median neurorrhaphy at the wrist or base of palm.

then turned into position. If frayed, they are cleanly cross-sectioned with a sharp knife. The wrist is held in a flexed position. The edge of the sheath of the proximal end of the median nerve, close to its little volar vessel, is pierced with a tiny curved needle and the silk drawn through. The needle is then passed through a homologous point in the sheath of the distal end. The sectioned surfaces are moistened with salt solution from a medicine dropper and the suture is carefully tied so as to approximate the little vessel. By this procedure the nerve is brought together without twisting. The stitch ends are clipped in a small hemostat which is used to steady the nerve while a second stitch is placed through the sheath on the opposite side of the vessel.* Before being tied, the nerve ends are again moistened with a few drops of saline. These stitches are also clipped in a snap which is used to rotate the nerve so that a stitch

* In order to avoid bruising the nerve while the first stitch is being placed, it may be found helpful to steady it gently with a saline soaked cotton pledget held in a snap.

may be placed on the side of the nerve and tied under saline drip. In this manner the circumference of the median nerve is approximated by five or six sutures. The epineurium should be nicely
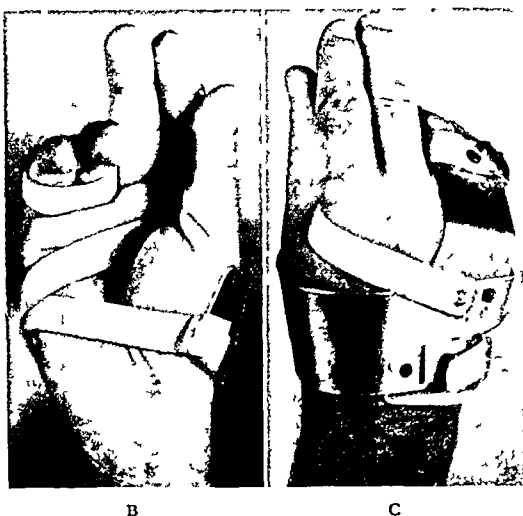

B   C

FIG. 11B AND C. The flanged cross hand splint in use. The metacarpophalangeal flange prevents undue stretching of the hypothenar muscles (ulnar). The thumb flange similarly protects the thenar muscles (median and ulnar). The medial flange steadies the hand in the splint. It is lined with felt or applied over a glove from which middle and distal phalanges have been cut. It is readily removable for washing.

closed. No gaps should be left unclosed. The ulnar nerve is similarly repaired, but twisting must be avoided as much as possible without the guide of a tiny vessel. The suture lines of the nerves are *not* wrapped in fat. Flexion of the wrist is still maintained while the palmaris longus tendon is sutured, the annular ligament and fascia approximated with fine catgut, while the skin is closed with interrupted silk, and until a splint is applied. No drainage is used. If the wound is so soiled that drainage is necessary, immediate neurorrhapy (and tenorrhaphy) should not be attempted. This is a safe rule.

Such a wound should be cleansed and Dakin's treatment instituted. The sheaths of the nerve ends may be caught together with a stitch. This facilitates subsequent repair which should be postponed for some weeks after complete wound healing has occurred. The splint should be worn for a couple of weeks to minimize the retraction of the tendons. Until the repair operation is undertaken, joint stiffness and the stretching of atrophying muscles must be combated.

At the Root of the Palm.   The term root of the palm is used to designate the interval between the proximal margin of the annular ligament and a line drawn on a level with the outstretched thumb. Many structures lie within this small compass. The author has seen injuries in this situation from falls on glass tumblers and bottles, a fall on a scythe, revolving blades of a wood fluting machine (Fig. 5) and porcelain faucet handles. Bones were transected in two instances. These accidents always present an exercise in anatomy and in surgical dexterity. Neurorrhaphy in this situation has been facilitated by magnifying lenses, by steadying the nerve with a saline soaked cotton pledget held in a snap, and by using tiny curved atraumatic needles.

In the Digits and on the Dorsum of the Hand.   The digital nerves in the fingers show a remarkable propensity for regeneration. Restoration of function may require a considerable period of time, but in the author's experience if the edges of a fresh wound be carefully approximated, regeneration practically always occurs. This obtains, of course, only in wounds which heal by first intention. The author also believes that the digital nerves in the palm possess this same aptitude. This has been demonstrated in a number of cases of deep, stellate lacerations where, although no attempt was made to suture the digital nerves, complete restoration occurred. Since, however, these nerves carry motor fibers to the lumbricals it is better practice, if conditions permit, to search for them and approximate them with one or two sutures. This is especially important in case of the index finger.

The rami of the sensory branch of the radial nerve on the dorsolateral aspect of the wrist and hand, as well as the dorsal cutaneous branch of the ulnar nerve, should be sought and sutured. In skilled laborers and musicians, if the acute injury is not recognized, subsequent suture will be necessary.

Late Neurorrhaphy.   Wide, delicate dissection through scar tissue is necessary for the identification and mobilization of structures. (Figs. 6 and 7.) A piece of rubber tubing to serve as a tourniquet is included in the kit. It is convenient to have at hand for temporary use in certain cases. In old ulnar nerve injuries at the wrist and root of the palm, for example, the vessels may have become converted into a plexus. A tourniquet, for a short time, greatly aids dissection and minimizes the trauma of sponging.*

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* The author still prefers a sterile rubber tourniquet to a blood pressure cuff beneath the sterile drapes.

All scar tissue between nerve (and tendon) ends must be removed and the ends themselves prepared for suture by clean transection with a sharp knife. The proximal end of a nerve is usually sought first because it can be recognized by its "bulb." As the distal end possesses no characteristic marking, its discovery is less simple. The bulb is amputated. Tiny sections of the nerve ends are removed until both present normal appearing surfaces. This must be done conscientiously. The funiculi will be seen to extrude from the sectioned end. Fear of creating a sizable gap between nerve ends is, the author believes, the commonest cause of failure. *Nerve regeneration is impossible through scarred ends, regardless of the perfection of their suturing.*[4] The author also believes that end-to-end approximation without tension or twisting must be effected and has little confidence in the use of tubes (fascia, sections of veins, arteries, etc.) to bridge defects. Except in reconstruction of wrists and hands which have sustained wide tissue loss (see below) flexing the hand and elbow will usually be sufficient to permit suture at the wrist without tension. If not, the incision should be extended proximally and the nerve carefully freed for an appropriate distance. (Fig. 7.) If approximation cannot then be effected, in case of the ulnar nerve, the nerve may be transplanted anteriorly at the elbow. This has worked beautifully in five cases.*

Every effort, indeed, should be made to accomplish end-to-end suture without tension or twisting because the likelihood of success of this method of nerve repair so greatly exceeds that of all others. By securing the most favorable position of the digits of the wrist and elbow to shorten the space between nerve ends, by freely mobilizing the proximal and distal ends of the nerve or by transposition of the ulnar nerve at the elbow, the author in a considerable experience has never found it necessary to resort to free nerve grafting in the hand and wrist. In other regions gradual nerve lengthening by a two-stage operation[6,8] may overcome a wide defect

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* In the December 1938 number of this Journal the author published his study of injuries to the hand, based upon 1100 personally treated cases, exclusive of minor cuts, abrasions, contusions, infections, burns and foreign bodies. The series included 138 cases of injury to nerves which form the basis of the present article. This recent analysis discloses five cases in which the ulnar nerve was transplanted anteriorly at the elbow to effect suture at the wrist instead of three as reported in the previous paper.[4] These are:

<div style="margin-left:3em">

R. R.   F 45 operated on Dec. 28, 1925   5 mos. after injury
H. W.   M 17 operated on Dec. 21, 1927 12 mos. after injury
N. D.   M 23 operated on May 21, 1931 14 mos. after injury
S. J. S.   M 19 operated on June 27, 1931 23 mos. after injury
E. L. S.   M 33 operated on Apr. 17 1933   5 mos. after injury

</div>

and obviate nerve grafting. The procedure has never been found necessary, however, in the hand and wrist.

In cases of maiming by old shotgun injuries or high voltage burns the correction of the contracture by some type of pedicle graft, later stabilization of the part by bone grafting, and the restoration of gross function by still later tendon grafting have so contented several patients that subsequent nerve grafting has not been desired. In multilating shotgun injuries to the hand, conservative surgery (thorough cleansing, debridement and dakinization, painstaking attention to the preservation of suppleness of the joints, followed by simple plastic procedures) has fortunately given functional recoveries exceeding primary expectation. Certain disabilities may be ameliorated by tendon transplantation and arthrodesis. Conditions requiring nerve grafting may, however, arise.* Although this procedure is viewed askance by some writers, successes have been reported in the field under consideration by several surgeons, notably Bunnell.[1,2] The use of a single section of nerve of the diameter of the injured nerve is preferable to several grafts of smaller diameter arranged as a cable. Careful closure of the epineurium at both ends is imperative. The graft serves, of course, merely as a conduit for the advancing neurofibrillae.

It has been suggested that the graft be sutured into the defect only at the proximal end and that it be somewhat longer than the defect. After a calculated interval of time the wound is reopened, the graft exposed and the distal end transected until the advancing neurofibrillae are encountered. The distal end of the injured nerve is then appropriately prepared and sutured to the lower end of the graft. The author has had no experience with this procedure. It is designed, of course, to thwart a possible scar tissue obstruction at the distal junction.

In cases with partial palsy in which improvement has not occurred following the use of appropriate measures to favor spontaneous regeneration, operation may disclose the nerve roughened to greater or lesser extent by fibrous adhesions, and perhaps somewhat thickened. This creates a problem of not infrequent occurrence. A decision must be made between neurolysis with or without endoneurolysis and resection with suture. The relative duration of the

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* The author has bridged a gap of 3½ inches in the common peroneal nerve by a graft from the sural nerve. There has been perfectly satisfactory regeneration through the deep peroneal nerve but not through the superficial peroneal nerve. This may be due to discrepancy in the size of nerve end graft. Apparently, however, no lateral neuroma has developed.

convalescences following the two procedures renders the decision still more difficult. The history as regards date and nature of the accident, the age and occupation of the patient, and the extent of the palsy may be determining factors. Information of only gross nature can be ascertained by electrical stimulation of the nerve proximal to the pathology. When in considerable doubt the author prefers neurolysis with or without endoneurolysis. If it succeeds much time has been saved. If it fails a resection can subsequently be performed. The use of undamaged contiguous muscle or pedunculated fat grafts to protect the nerve from further adhesions is rarely practicable in the wrist and hand. Free fat is of doubtful benefit. The use of a membrane for this purpose has been too infrequent to warrant judgment as to its merit.

When a *lateral neuroma* is removed, the naked area should be covered with epineurium. To effect this without constriction of the nerve, a pedunculated strip of sheath may be carefully raised from the neuroma. After the clump of neurofibrillae entering into the bulb have been drawn tense and divided, the flap is caught over the defect with a couple of tiny silk sutures.

Splinting. After every neurorrhaphy a splint should be used. If the sutured nerve contains motor fibers, the splint should serve three purposes: (1) It should prevent tension on the suture-line to facilitate regeneration. (2) It should protect the muscles supplied by the injured nerve from being stretched by the pull of antagonistic muscles. (3) It should leave free for active use as much of the hand as possible. To prevent tension on the suture, the splint, as a general rule, should maintain the part in the position in which the suture was effected. If, for example, it was found necessary at operation to flex both the wrist and elbow to effect the suture without tension, the splint should maintain this position. This may be accomplished with a moulded plaster gutter. For immediate neurorrhaphy at the wrist a device for maintaining flexion of the wrist alone is usually sufficient. A simple, felt-covered aluminum splint* with appropriately placed buckle straps has been found satisfactory. (Fig. 8.) This splint also permits the early use of tendons which must often be coincidently repaired. By slightly angulating the splint at the knuckles, strain on the hypothenar muscles, the lumbricals, and the palmar interossei may be relieved. With early gross movements of the fingers this second angulation at the knuckles may not be deemed necessary. It may, moreover, have the disadvantage of

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* For old injuries, in which deviation of the fingers has occurred, side flanges, with or without interdigital flanges, may be advantageously added.

straining the dorsal interossei in ulnar nerve injury. In connection
with the wrist splint the author uses a suede thumb-cot. (Fig. 9.)
This simple device effectively protects the atrophying superficial
thenar muscles in case of median nerve injury and the deep muscles
in case of ulnar injury.

This combination of wrist-splint and thumb-cot (Fig. 10) is worn
for two months. If it is taken off occasionally by the surgeon and
the wrist and thumb gently moved, no stiffness will occur. After this
lapse of time nerve regeneration should have sufficiently progressed
to withstand tension at the wrist. The second and third desiderata
still obtain to prevent the stretching of atrophied muscles and to
leave as much of the hand as possible free for exercise. In case of
median neurorrhaphy use of the thumb-cot alone is then continued
for some weeks. During this period the patient is supervised in
exercises in apposition and abduction of the thumb and in extension
of the middle and distal phalanges of the index finger. The time
when the patient may be permitted to remove the cot himself for
these exercises is determined, of course, by his intelligence and his
spirit of cooperation. Although the thumb-cot does not support the
lateral lumbrical muscle, the author has not seen any permanent
impairment of extension of the middle and distal phalanges of the
index finger.

After removal of the wrist splint and thumb-cot combination in
case of *ulnar neurorrhaphy* or of both ulnar and median neuror-
rhaphy, the author uses a flanged-cross hand splint. (Fig. 11.) This
splint is worn for twelve or sixteen weeks. During this period if the
ulnar nerve alone has been sutured, the patient is supervised in
exercises in abduction and adduction of the fingers, flexion of the
fingers as rods at the metacarpophalangeal joints, extension of the
middle and distal phalanges and in adduction of the thumb. If
both median and ulnar nerves have been sutured, exercises in appo-
sition and abduction of the thumb and in extension of the middle and
distal phalanges of the index finger are added. As the muscles
develop and the breadth of the hand increases, a similar but wider
splint may be required. Here, again, the time when the patient may
be permitted to remove the splint himself for exercises is determined
by his intelligence and his spirit of cooperation. Although the
flanged-cross hand splint does not definitely support the first,
second, and third dorsal interossei, the first palmar interosseus, and
the lateral lumbricals, the author has seen no permanent impair-
ment of function of these muscles. This happy circumstance may
be attributable to personal supervision. He has occasionally been

disappointed by some persistent weakness of the third palmar interosseus, as though it had been stretched by the more powerful abductor minimi digiti, leaving a slight interval between the ring and little fingers. This might be obviated by providing the metacarpophalangeal flange with a medial flange instead of depending wholly upon a buckle strap. (Fig. 11B.) The author has never been aware of any functional impairment attributable to pressure of the buckle straps. The straps, of course, must not be too tightly applied. On the contrary, as noted above, as a hand increases in breadth, a similar but wider splint becomes necessary.

After-Care. The more closely the operative technique approaches the ideal, the greater will be the likelihood of restoration of function. The postoperative course, however, cannot be left to chance. Quite as important as the operation is painstaking attention to details in the after-care. This cannot be too strongly emphasized. The convalescence is long. Thorough periodic examinations are necessary for several reasons: to detect the slightest evidence of progress, to determine the changing needs of muscle support and muscle exercise, to maintain the morale of the patient and to stimulate his coöperation. There can be no fixed rules. Each individual is a law unto himself.

Although nerve regeneration may be expected at the rate of a millimeter a day, the speed of regeneration may vary in the different branches of a given nerve. In following the progress of regeneration of a mixed nerve, motor tests are more reliable than sensory tests. Effort should be made to detect contraction in the muscle bellies and to avoid the deception of supplementary motility. The interpretation of tests for protopathic and epicritic sensibility should be conservative. The pitfalls of sensory overlap must be avoided. Tinel's sign has been found very suggestive especially when performed as a series of blunt pressures along the course of the nerve rather than by percussion.

At each visit the splint is removed and with the hand and wrist appropriately supported all of the muscles are tested individually and coördinately. The mobility of joints is maintained, if necessary, by massage and gentle passive motions. In due time the patient is permitted to remove his splint several times a day to perform certain exercises. At each subsequent visit special instructions are then given. Later he is encouraged to perform small tasks (with light tools, nails, screws, practicing on a musical instrument). Although in other regions of the body electrical stimulation of atrophied muscles is practiced, the author does not employ it in the hand.

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DISCUSSION

HENRY C. MARBLE (Boston): It is needless for me to comment upon this work that Dr. Harmer has told us about. It is a beautiful exercise in anatomy and surgical technique, and the results he showed here speak for themselves.

There is one question I want to ask him that he did not take up. Perhaps it isn't pertinent. How does he handle gross defects of nerves, such as an inch and a half or 2 inches lost from the median nerve?

There is one other phase that interests me just at the moment, and that is in the matter of painful scars and neuromata. In the past we have made an effort to prevent them by injection into the nerve with alcohol. The results have not been particularly satisfactory. For about two years I have been injecting these nerves with ether. The pathologists tell me that ether has one advantage. It dissolves the myelin sheaths very completely and if this is done, the neuromata will be less painful and less incapacitated. My personal experience has been that the anesthesia produced from injecting ether into the nerves is complete and prolonged.

TORR W. HARMER (closing): I wish to thank Dr. Marble for his discussion of my paper. In order to bring the discussion of the paper within the allotted time, I have deleted a number of passages which will appear in the printed article. These answer in detail the first and last questions which Doctor Marble asked. In view of the lateness of the hour I pray you and he will excuse me if I refer you to the published paper.

I may state briefly that, as you all know, free mobilization of the proximal and distal ends of the median nerve combined with flexion of the wrist, fingers and elbow will on most cases admit end-to-end suture without undue tension. In wider defects gradual lengthening with secondary suture, as suggested by Naffziger, Pollock and Davis, may be practiced. The technique of nerve grafting is too long a subject to discuss here. Suffice it to say that a free graft of the diameter of the injured nerve is much more likely to serve as a satisfactory conduit for the advancing neurofibrillae than a cable of several sections of lesser diameter. The graft may be sutured to both ends of the defect or a section longer than the defect may be sutured first at the proximal end and, after a calculated interval of time, suture at the distal end may be completed.

I am interested in Dr. Marble's remarks about smelling his novocaine before he injects it and about using ether instead of alcohol for the injection of neuromata in amputation stumps. In the treatment of terminal neuromata I practice excision, with section of the nerve well above the bulb. In finger stumps this may be fussy work but can usually be satisfactorily accomplished under novocaine or evipal with the aid of magnifying lenses which clasp over spectacles, as shown in the first illustration. I shall be interested to try the injection of ether instead of alcohol in cases of causalgia.

In regard to Dr. Marble's last question: It is my understanding that the extensor tendons of the fingers with their muscle bellies supplied by the radial nerve extend the fingers at the metacarpophalangeal joints, but that the burden of the extension at the middle and distal joints falls upon the interossei supplied by the ulnar nerve and the lumbricals, the two lateral supplied by the median and the two medial supplied by the ulnar. Extension of the middle and distal phalanges of the index finger is accomplished, I believe, wholly by the first lumbrical, despite the two extensor tendons. The middle finger possesses two interossei and one lumbrical and the ring and little fingers one each, with, of course, two extensor tendons in the case of the little finger. The third lumbrical going to the ring finger has often a double nerve supply (median and ulnar), and there may be an anastomosis between the deep branch of the ulnar and the median at the root of the palm.

KNEE INJURIES INCIDENT TO SPORTS AND RECREATION

AUGUSTUS THORNDIKE, JR., M.D., F.A.C.S.

BOSTON, MASSACHUSETTS

IN addressing this association whose main interest is trauma, it occurred to the author that perhaps to date too little interest has been devoted to trauma produced in sports and recreation. Most of our medical colleagues caring for traumatic injuries see mainly those cases that arrive in our hospitals with the more serious traumatic lesions. There is, however, a large and ever-increasing field of lesser and yet important trauma occurring among those individuals who enjoy sports and recreation.

The interest in sports among old and young is worldwide. It has been estimated that over eight million participate in school, college and professionally organized sports in this country alone. Furthermore, over a million sport lovers indulge informally in gold, tennis, mountain climbing and skiing. Frank Mencke[1] informs us that the interest of followers or spectators in organized sports in the United States reached these remarkable totals last year: basketball, ninety million; softball, seventy-two million; baseball, sixty million; football, forty-five million. Nor is this peculiar to our own country! A recent Associated Press dispatch recorded 150,000 spectators at the championship soccer game between England and Scotland.

Most of these players experience some injury, usually minor, but occasionally serious in nature. Our interest has not been focused sufficiently on simple strains, sprains and contusions. Furthermore, in general, the treatment such injuries receive at the present time is inadequate. Today knee joint traumata have been selected to emphasize adequate treatment of all types of injuries, in order that recurrent or additional more serious injury may be prevented later.

When trauma to this joint is seen within a few seconds of the time of injury, rather than an hour or more later, there are certain definite points concerning diagnosis and treatment that might be of interest. There are few of us who have the opportunity of seeing this particular phase. It is of further interest in that adequate treatment promptly instituted will benefit the patient.

To understand the results of trauma to this joint, a thorough knowledge of the pathologic picture is essential. Little emphasis has been placed on this, but much should be. If either a joint or bursa

contusion, muscle (vastus medialis) contusion, sprain, or partial luxation of the joint surface, with a tear or loosening of the menisci and capsule takes place, certain definite events follow in quick order. In each and every one of these types of injury, there is always concomitant hemorrhage from torn areolar tissue, blood vessels, and capillaries, whether within or without the synovial membrane. Hemorrhage promptly takes place, in addition to the tearing of specialized tissue. During the process of repair, one expects in sequence hematoma formation, hematoma absorption and healing by fibroblastic repair. Such, then, in brief is the pathology of the trauma and its repair. The problem is to estimate promptly the extent of the lesion or lesions and institute immediately the appropriate and adequate measures to control and protect first the tissues torn, and second the hemorrhage and hematoma.

The diagnosis of the contusions, sprains, and possible internal derangements at the time of injury is difficult. The picture is utterly different from that seen one hour or more afterwards! There is but one way to proceed with the examination. First, palpate the bony prominences, the areas over the vasti muscles and over the ligaments, to elicit points of tenderness, if any. Second, with the muscles relaxed, test for ligament weakness by gentle manipulation, searching for abnormal lateral, medial or anteroposterior mobility. And third, test active muscular function for flexion and extension. Three such points taken in sequence will reveal a provisional diagnosis. It is not wise to give a final diagnosis until the patient is seen the following day, after the first and most important phase of the treatment has been carried out. Then repeating the three steps of the examination, a more accurate diagnosis both as to type of injury and degree of injury and probable prognosis as to period of convalescence can be made. Reserve prognostication always until twenty-four hours after such an injury!

It is needless to describe the accurate points of palpation and the function tests to be carried out to diagnose either a sprain of the medial collateral ligament or a joint capsule contusion or a meniscus injury. They are known by all surgeons interested in trauma. The frequency with which these different types of injuries occur in organized sports is given in Table 1. The striking feature, I believe, is the low incidence of internal derangements among these 483 cases (less than 10 per cent). This is attributed to treatment and especially to subsequent prophylactic treatment. A firm conviction that menisci are rarely injured with the first sprain of a knee holds the attention of my entire staff. To be sure, it occasionally happens when

the trauma is severe enough to tear the joint capsule, but this is most unusual and accounts for only three cases (one of these never

TABLE I

KNEE JOINT INJURIES IN ORGANIZED SPORTS OVER A SEVEN YEAR PERIOD AT HARVARD UNIVERSITY

(Exclusive of lacerations and other less serious injuries)

Ligament sprains	203
Joint contusions	145
Muscle contusions	78
*Internal derangements	43
Bursa contusions	8
Superficial contusions	6
	——
Total	483

* Inclusive of all meniscus and crucial ligament injuries.

developed a meniscus injury, in spite of the capsule tear). These sprains are analyzed in Table II to show the particular ligaments involved, in a series of 246 sprained knees.

TABLE II

KNEE SPRAINS IN ORGANIZED SPORTS AT HARVARD UNIVERSITY

Ligaments Involved	1932–1933	1933–1934	1934–1935	1935–1936	1936–1937	1937–1938	1938–1939	Totals
Medial collateral ligament	37	21	26	16	18	20	11	149
Lateral collateral ligament	4	2	7	8	4	7	0	32
Crucial ligament	4	4	2	0	0	3	0	13
Medial collateral ligament and medial meniscus	5	8	2	7	0	0	1	23
Medial collateral and lateral collateral ligaments	0	2	0	1	2	1	0	6
Medial collateral ligament and lateral meniscus	0	0	0	1	0	0	0	1
Medial collateral and crucial ligaments	0	1	0	3	0	1	0	5
Lateral collateral and crucial ligaments	0	0	0	0	0	0	0	0
Lateral collateral ligament and medial meniscus	0	0	0	0	0	0	0	0
Lateral collateral ligament and lateral meniscus	0	0	1	0	0	0	0	1
Tibiofibular ligament superior	0	3	0	0	0	0	0	3
Patellar ligament	0	1	0	0	0	0	0	1
No ligaments mentioned	4	3	2	1	0	2	0	12
Totals	54	45	40	37	25	34	12	246

The total individual ligament sprains are recapitulated in Table III.

The treatment of knee injuries divided itself into three different periods: (1) Early treatment, (2) convalescent treatment, and (3) prophylactic treatment to prevent a recurrence of the injury. Rarely is the opportunity presented to see these injuries immediately after they take place, the optimum moment to start treatment.

Recalling the pathology of contusions and sprains, the immediate treatment is aimed toward the control of hemorrhage and the

TABLE III

LIGAMENTS INVOLVED IN KNEE SPRAINS

Medial collateral ligament	149
Lateral collateral ligament	32
Medial collateral ligament and medial meniscus	23
Crucial ligament	13
Medial collateral ligament and lateral collateral ligament	6
Medial collateral ligament and crucial ligament	5
Tibiofibular ligament, superior	3
Medial collateral ligament and lateral meniscus	1
Lateral collateral ligament and lateral meniscus	1
Patellar ligament	1
Unspecified	12
Total	246

minimizing of the hematoma. There are only three steps to this: (1) the application of cold, preferably ice water for one hour, followed by (2) the application of a compression bandage, utilizing sponge rubber, and (3) rest. These are the immediate measures to control subsequent fluid or blood accumulation in or about the joint. When these measures are carried out promptly, one rarely sees "water on the knee" as an accompanying complication in sprains and contusions. The sponge rubber compression yields good results. When the bandage is first removed, twenty-four hours after its application, ecchymosis is evident at the margin of the rubber pads. This represents deep hemorrhage controlled and forced by compression from the deeper tissues into the skin. Once brought to the superficial tissues, its absorption can be more effectively treated by massage. The hematoma is dispensed with, and there is little or no fluid in the joint. The joint capsule is not distended the day following the injury. In the most severe primary injuries with tears of the ligament and capsule, when more than 10 degrees of abnormal mobility is elicited in the first examination, the early treatment is instituted as usual. However, a light cylindrical cast is applied outside the compression bandage, and the patient is hospitalized with the leg elevated on three pillows for forty-eight hours. Even in such cases joint effusions are exceedingly rare.

The convalescent treatment is aimed to promote the absorption of the hematoma and waste products of hemorrhage and to hasten the tissue repair. The two agents resorted to for stimulation of
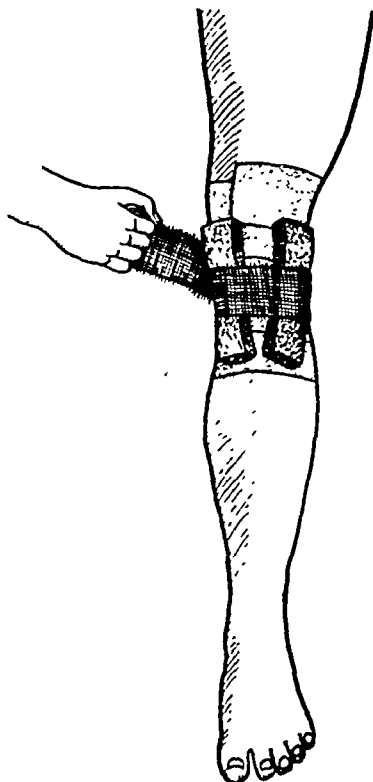

FIG. 1. The use of sponge rubber in a compression bandage of the knee. (From Thorndike's "Athletic Injuries," Lea & Febiger.)

lymphatic absorption are *heat* and *massage*. At first these are applied proximal to the site of trauma, and at about the fifth to seventh day actually on this site. Vigorous massage is always contraindicated, and only gentle stroking and effleurage should be tolerated. Between treatments, the sponge rubber compression bandage is worn, and weight bearing and physical activity gradually permitted. No longer is joint effusion a complicating factor, and no longer is withdrawal of fluid by needle and syringe necessary.

The patient is not permitted to return to sports or recreation until muscle tone has returned to normal and until all tenderness in the ligaments or contused tissues involved has disappeared, and then only with a restrictive adhesive strapping and padding to prevent

FIG. 2. Ecchymosis visible in the skin twenty-
four hours after the injury at the margin of
the rubber pad.
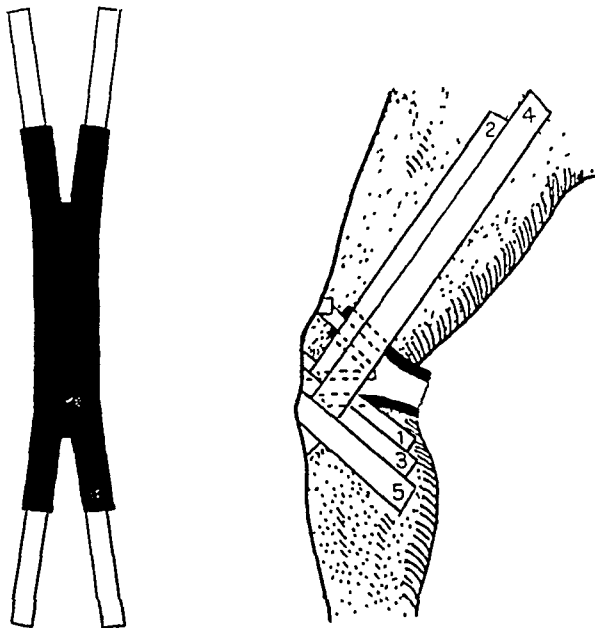
FIG. 3. Basic principles of the Duke Simpson knee strap-
ping. (From Thorndike's "Athletic Injuries," Lea &
Febiger.)

recurrent injury. This prophylactic period of treatment is most important and should continue always, particularly in sprains. Ligamentous tissues heal by fibroblastic repair, always remain weak or "brittle," and cannot withstand the stress or strain of a normal ligament. "Once a sprain, always a sprain."[2] The prophylactic strapping is indicated from then on in future years. It is more frequently in the recurrent sprain that the meniscus and other internal structures of the knee are injured. Many high school boys with sprained knees have been carried through a college athletic career without recurrent sprains by careful adherence to this régime. Many a serious internal derangement of the knee has been prevented.

For the last seven years, with ever-increasing success, this method of treatment has been carried out in a series of 246 knee sprains. The results are gratifying; the recurrent injuries and the more serious internal derangements of the knee are becoming less frequent. Last autumn on the Harvard varsity football squad of thirty-five men, there were sixteen knees previously sprained that were prophylactically treated daily for eleven weeks of play. Only one of these experienced a recurrent injury, and that a mild one. The steady decline in the number of more serious injuries proves the value of any treatment.

TABLE IV

RATIO OF SERIOUS KNEE INJURIES IN ORGANIZED SPORTS AT HARVARD COLLEGE

	1932–1935, Incl.		1935–1938, Incl.		1938–1939*	
	No.	Ratio	No.	Ratio	No.	Ratio
Knee sprains	110	11	77	8	11	11
Internal derangements	29	3	19	2	1	1

* Through April, 1939.

CONCLUSIONS

1. The adequate treatment of knee injuries incident to sports and recreation requires the institution of early and prompt diagnosis, and immediate application of cold, compression bandage, and rest, to control hemorrhage and protect the injured tissues.

2. During the convalescent treatment, measures are instituted to promote the absorption of the waste products of hemorrhage, and to stimulate tissue repair.

3. All former sprains should be protected by adhesive strapping to prevent a recurrence of such injuries in sports or recreation where stress and strain are expected.

4. The steady decline in the ratio of serious internal derangements of the knee joint among knee sprains in organized athletics is proof that this treatment is of value.

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DISCUSSION

HENRY H. RITTER (New York City): It was a great privilege to listen to Dr. Thorndike's very able discussion of knee joint traumata in sports and recreation. He has an opportunity that is afforded very few of us, of seeing these accidents immediately after they occur, of treating them immediately, and following them through their entire course. The results he has shown are evidences of intelligent, intensive, and early treatment. I envy him his position.

The cases of sport injuries to the knee that come to me usually appear some time after the occurrence of the accident and are mostly in the older people than those seen by Dr. Thorndike. The majority are men in the employ of the larger corporations which further sports among their employees, or in children from the city playgrouns. The games are baseball, basketball, bowling, and, to a lesser extent, football. When the patients get to me, as a rule, the knee joint is swollen and there is fluid within the joint, but I rarely ever see them until many hours or days after the accident.

Dr. Thorndike has shown us the marvelous reduction in the number of sprains to the knee joint in football accidents. I did not quite understand whether that was because of the pre-sport examination of candidates for the teams or whether it was the result of excellent coaching or splendid training, or a combination of all three.

Our percentage of injuries to the meniscus, and particularly the medial meniscus, is somewhat higher than his. That may be the result of dealing with older people who work all week, are not well coached and not well trained, or due to the fact that we do not have all accident cases reported if they are apparently of a minor nature. Dr. Thorndike's number is extremely small.

We agree that the medial meniscus is the most commonly damaged; we agree that the median collateral ligament is the most commonly involved.

Recently a man who is associated with one of the larger teams told me that no matter how trivial they are, he immobilizes knee joint injuries for a period of two or three weeks. He starts with passive motion, but I mention that only to condemn it. I see nothing passive about passive motion except the name. There is nothing passive about a doctor manipulating a painful knee while the patient screams and yells in pain. Any time such a thing as passive motion is necessary, I think it should be done under anesthesia.

A patient of mine, a girl 13 years of age, who was on the track team of a boarding school, was high jumping when she injured her knee. Forty-eight hours later, she was brought to me. The knee joint was swollen, painful, and tender. A small body was noted by x-ray in the intercondyloid notch, that piece of bone that comes from the articular surface of the lateral condyle of the femur. It was a fracture of the cartilage and bone.

The patient was sent to the hospital and prepared. On the following morning, through a lateral incision, I opened the knee joint and found the joint full of blood which was evacuated and we removed the specimen. We immediately had her move the knee, the afternoon of the same day. On the fifth day, she was out of bed and on the seventh day, she was walking about. Two weeks after her operation she could keep the knee completely extended. She engaged in sports three momths after the operation.

In another sports accident, the patient was a rather stout lady who wanted to do some skating to keep down her weight. She fell and sustained an anterior dislocation of the leg which was very easy to reduce. She had no restraining apparatus and no cast applied. She developed pneumonia on the afternoon of the second day, stayed in bed for some weeks, and when she got up, she was able to bend the knee to a right angle and extend it to a straight angle.

A young lad who played on the baseball team of one of the gas companies in our state had had an accident eight months before. He was sliding into base, and his knee locked on him. On the following day, I saw him. A foreign body could be observed in the x-ray and a depression in the condyle from which the body came. The diagnosis was osteochondritis dissecans. I did not think it had anything to do with the accident. During the examination, I found that the opposite knee had been operated upon. We got in touch with the hospital where the operation was performed and they sent us all the radiographs made at that time of the operation. An x-ray taken eight months before the alleged accident showed a typical osteochondritis dissecans, not separated. We removed the loose body. There was no hemorrhage. It was perfectly smooth and was not bleeding. There was no evidence of trauma.

EDGAR L. GILCREEST (San Francisco): The thing that interested me most was Dr. Thorndike's statistics showing that the ligaments were injured the greatest number of times, the joint surfaces second, and the muscles third.

I have been particularly interested in these lesions of muscles for fifteen years. I have seen many patients with muscle tears who have gone for a considerable length of time, having pain referred to their joints, and who were given various forms of treatment to the joint itself, but when the muscle was repaired, they would become well and would return to work 100 per cent recovered, often within three months after the operation. I

would like to ask Dr. Thorndike, if he has found many of these lesions of muscles near the joint?

AUGUSTUS THORNDIKE, JR. (closing): I wish to leave you with the impression that these statistics were taken from all sports and not only from football. The reduction in the number of sprains, of course, is partly due to better training. It is also due to the fact that we now strap all the formerly sprained knees. At the beginning of this period, those boys who were taking part in the intramural athletic program (not intercollegiate), were not strapped. Now, however, they receive exactly the same treatment and care as our varsity. Therefore, more people are being protected against recurrent sprains than used to be.

The question of immobilization of knees—the only knee injuries we immobilize are those presenting 10 degrees abnormal mobility or more, or a tear of the capsule. We do immobilize them forty-eight hours in bed with the leg elevated. We split the cast after forty-eight years, and find no effusion in the joint. We start them on crutches and gradually make them walk and give them physiotherapy beginning the following day.

I do not believe that passive motion or forceful motion should ever be attempted on this type of injury.

Osteochondritis is a very interesting thing. We have not seen a single instance of it in boys of college age. I know it happens, but they just have not drifted into our clinic.

As to Dr. Gilcreest's question on lesions of the muscle, I take it he means a tear of the quadriceps fascia and a muscle herniation following it. The pain may be referred to the knee. We have had none of those in the thigh since I have been responsible to the athletic teams, but I know of several men my age who received such injuries in college football, where the fascia was torn over the quadriceps and the muscle protruded through the fascial tear, presenting a true muscle hernia. I have seen it, however, in the lower leg in sprinters and hurdlers, causing a good deal of discomfort. Repair, of course, relieves the symptoms.

A TOPOGRAPHIC ATLAS FOR X-RAY THERAPY. By Ira Kaplan, M.D. and Sidney Rubenfeld, M.D. Chicago, 1939. Year Book Publishers, Inc. Price $5.00.

With the modern advances in the use of radiation therapy, particularly in malignant disease, but also in a wide variety of less serious lesions, a guide for the therapist to the anatomic landmarks of internal structures is a manifest necessity. Dr. Kaplan, head of the department of radiation therapy at Bellevue Hospital in New York and director of the division of cancer of the New York City Department of Hospitals, has, with the collaboration of Dr. Sidney Rubenfeld, now presented such a tpoographic atlas.

The book consists of a series of plates, showing the projection of internal organs on the body surface, their relationships to visible and palpable landmarks, and the proper situation of radiation cones for treatment directed to these organs. The explanatory text gives suggestions for placing the cones with regard to the external features, and describes the direction they are to take for particular therapeutic results. A particularly valuable feature is a series of plates showing the lymphatic drainage areas associated with those organs most commonly affected by cancer.

The drawings, excellent work by Evelyn Madsen, are clear and distinct, and are likely to be more helpful to the therapist than the x-ray films usually presented.

BEESLY AND JOHNSTON'S MANUAL OF SURGICAL ANATOMY. Revised by John Bruce, F.R.C.S. and Robert Walmsley, M.D. Fifth Edition. London, 1939. Oxford University Press. Price $6.50.

When the first edition of this work came out in 1918 it received excellent reviews and enjoyed a wide distribution. In this, the fifth edition, the entire text has been thoroughly revised, some new matter introduced and accounts of several surgical procedures which are no longer practiced omitted. The book is divided into six sections, covering the head and neck, upper extremities, thorax, abdomen and pelvis, lower limbs, and vertebral column. The illustrations (187) are above average, and the index is good. A book that goes into a fifth edition speaks for itself.

DISEASES OF THE FOOT. By EMIL D. W. HAUSER, M.D. Philadelphia, 1939. Saunders. Price $6.00.

Dr. Hauser has studied the foot from all angles and has written a thorough study based for the most part on his personal experience, with methods tried in practice. Since 1922, when Professor Richard Scammon placed at his disposal a large series of valuable fetal, newborn and children's feet for anatomic study, his work has continued. At the Mayo Clinic, the large volume of material in the orthopedic department gave an opportunity to study the diagnosis and methods of treatment of diseases and injuries of the foot. A year as American-Scandinavian Fellow afforded him the privilege of studying under Professor Patril Haglund in Stockholm, whose work on clubfoot and infantile paralysis is classic. Dr. T. P. Noble imparted to him the teachings of his chief, Sir Robert Jones. From this long training and experience the present book stems. The subject is thoroughly covered. The illustrations are good. It is interesting. Since foot ailments are a common and important source of physical disability, this book can be read or studied with profit by both the general practitioner and the specialist.

ROENTGEN TECHNIQUE. By Clyde McNeill, M.D. Springfield, 1939. Charles C. Thomas. Price $5.00.

Dr. McNeill has given us a technical book of interest to those concerned with the general subject of roentgenology. The illustrations are excellent and number 268. The book is well arranged and full of suggestions and information. Although there are many good books on this subject, this one may be recommended for its time saving qualities.

EDITORIAL

SURGICAL RESIDENCIES AND APPROVED SPECIALIS'

AFTER 1941–1942 no physician will be eligible to take the examinations held by some of the American Boards for certification in a specialty unless he has held for a prescribed time a residency approved by the American Medical Association. Many physicians, mainly the younger group, claim that there are not sufficient residencies in this country to meet the demand, and that many capable men are therefore denied the opportunity to equip themselves for specialization.

For a long time we, too, had the same impression. In fact, we went further in our speculation—and thought that possibly in ten or twenty years, if only certified men could be specialists, the country would be faced with a shortage of competently trained men; there might not be enough recognized specialists in surgery (in all its branches) to administer to the country's needs.

However, the August 26, 1939 number of the *Journal of the American Medical Association* lists the various residencies and fellowships in approved hospitals or services in the United States, and the list shows that there is no apparent shortage. Residencies in specialties, as defined by the Council, are straight serv-

ices of one or more years, follow approved internship. We comput approved residencies in surgery, cology, and obstetrics and add combined residencies in obstetri gynecology. Our figures may be in error, but not enough so to aff conclusions.

In surgery, 303 physicians wei dents in August, 1939; in gyne there were twenty-one; in obstetr total was seventy-four; and in th bined field of obstetrics and gyne the number was 127. Therefore, as that this number will not decrea probability is that it will increas year to year as further facilities ar available), each year about 300 m be eligible to take their American examinations in surgery, and abc in obstetrics and gynecology. Whe numbers are multiplied by ten or 1 we arrive at the total of certified the end of ten or twenty years, wit allowance for decreases due to d retirement. It is apparent that th be no dearth of specialists in this c With the constant addition of af residencies as the years pass, the o may be the fact.

Every young man in training as an intern cannot be successful in getting a residency—not, at any rate, at the present time. Every physician who has the desire or inclination cannot become a certified specialist. Which may be a good thing. Otherwise, imagine the state of the profession if the United States had 120,000 specialists and a mere handful of general practitioners!

A grave responsibility rests on the shoulders of the surgical directors who select residents for the available places. They must make sure that the men they choose have the training, aptitude, possible manual dexterity, and moral fiber for the job. The right man should not be denied because those in power make wrong or poor selections.

T. S. W.

PERIPHERAL NERVE INJURIES, WITH RESULTS OF EARLY AND DELAYED SUTURE

C. R. G. FORRESTER, M.D., F.A.C.S.

CHICAGO, ILLINOIS

INTRODUCTION

IN the present day, with the increase in highway and industrial accidents, the subject of treatment of peripheral nerve injuries is of great interest to all surgeons. During the World War I was very fortunate to be associated with the British Army and was assigned to Alder Hey Military Hospital, Liverpool, where I had the great pleasure of working with Mr. T. R. Armour, F.R.C.S. and Sir Robert Jones for some seventeen months. Ultimately I taught this work of handling nerve injuries in that hospital. Little of a practical nature has been written on the subject of the treatment of nerve injuries, and for that reason, based on my experience during the War and in civil practice, I feel it advisable to discuss this important matter.

GENERAL CONSIDERATIONS

When we first went into the surgery of peripheral nerve injuries during the War we were of the opinion that a sensory nerve could be made to bridge a motor gap; in other words, that a gap in the ulnar, median or radial at or distal to the elbow could be bridged by using the musculocutaneous; likewise that the sciatic, posterior tibial, anterior tibial and peroneal could be bridged by using the long saphenous. After many attempts along this line we found that the interposed sensory nerve did not become converted into a func-

tioning bridge in a motor nerve. Unde those circumstances, we had to devis other means of overcoming our difficultie in nerve suture.

We found that end-to-end suture gav the only good results and that we mus suture only the sheath (not invading th neurons with suture material); that nerv suture must be done *without tension* or i would pull apart with disastrous results that a nerve gap could be closed by stretch ing the proximal or distal portion of th severed nerve (or both), to break u perineural adhesions, and by flexing th adjacent joints to shorten the nerve path that generally speaking the sutured nerv must be kept in this protected and relaxe position by a cast or other fixation for period of *forty-five days* after suture. W could then extend the flexed joint graduall over a period of two to three weeks or eve longer, a few degrees at a time, until th joint could be brought to normal positior thus allowing a gradual *stretching of th* healed nerve. We learned that the sensor fibers (of mixed sensory and motor nerve like the radial, median and ulnar) are th first to show unmistakable signs of re generation following nerve suture. The voluntary muscle control appears, and las appears the positive reaction of the reinner vated muscle to faradism (as tested by th Bristow coil); in other words, the proces of mixed nerve regeneration is (1) return o sensation; (2) voluntary control; (3) reac tion to faradism, in that order.

I wish to emphasize that to do nerve surgery, as well as to diagnose nerve lesions correctly, one must know the

BUTTONHOOK FOR
LIFTING   NERVE
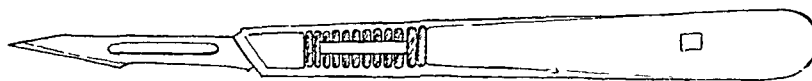
KNIFE FOR NERVE
DISSECTION –
FIG. 1.

anatomy intimately, or else decline to undertake this delicate and important form of surgery. I am not going into the detailed anatomy in the following regional comments, but I assume that the reader will study this in his own time and at his discretion.

In the *diagnosis of peripheral nerve lesions, and there only*, faradism is of use; it cannot be used for treatment after nerve suture. Sinusoidal galvanism is of great use, however, in the treatment of the affected muscles after nerve suture, but is of *no use* in diagnosis of nerve lesions. In other words, if before operation you wish to discover whether a nerve is severed, partially or completely, you use faradic stimulation above the site of the nerve lesion, over the muscles supplied by the nerve, and depending upon the strength of the response to this electrical stimulus you are able to determine the extent of nerve injury, whether partial or complete. In addition, you may map out the area of sensory impairment in testing for touch, pain (pinprick), heat and cold, and test the voluntary muscle actions. You may also note any muscle atrophy and any trophic changes in the member, to complete your diagnosis. If you are not conversant with peripheral nerve injuries, the above mentioned tests and the detailed anatomy, you should call in a surgeon or neurologist familiar with this type of work.

SURGICAL TECHNIQUE

In incising over the nerve lesion (new or old) I advise you to make your incision in line with the course of the nerve, and the incision should give a generous and sensible exposure of both the proximal and the distal ends. It is always good judgment to pick up the proximal nerve (normal, well above the wound or scar) and after having identified it, dissect distally to the point of nerve severance, likewise the distal end. This rule will save much trouble and should be applied to new and especially to old cases of nerve trauma, because by exposing and following the normal appearing nerve tract above and below the lesion, you are in a position to differentiate the abnormal or pathologic nerve tissue from the normal, as you dissect.

Do not keep at one end too long at a time, but change back and forth between

the proximal and the distal. This will avoid fatigue and give you a change of visual field and avoid confusion of structures.

the nerve ends, then *suture the sheath only*, using the very finest of needles and a double or triple zero catgut. (Fig. 2.) Use
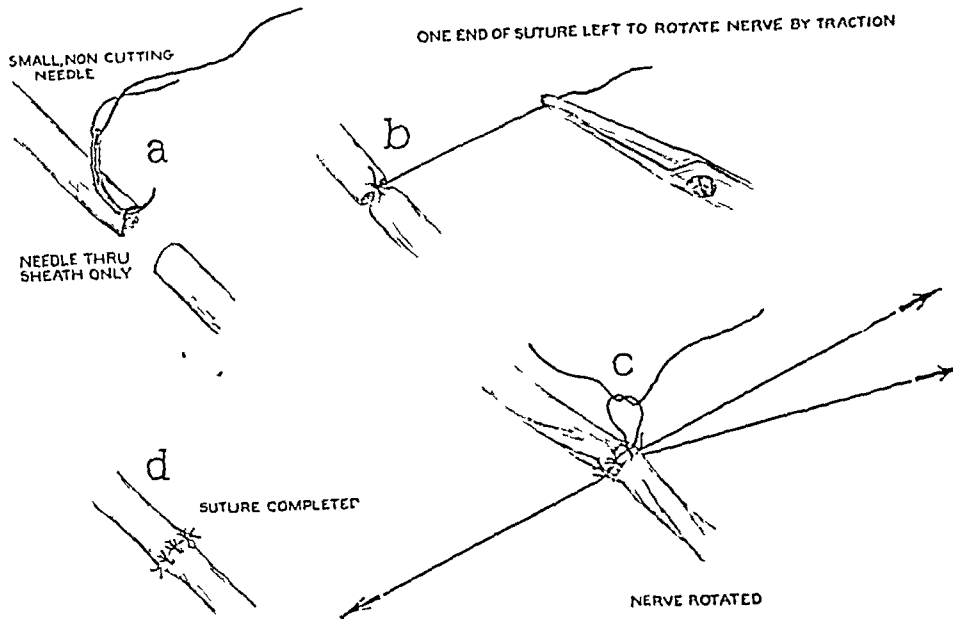

SMALL, NON CUTTING NEEDLE

ONE END OF SUTURE LEFT TO ROTATE NERVE BY TRACTION

NEEDLE THRU SHEATH ONLY

SUTURE COMPLETED

NERVE ROTATED

FIG. 2.

Peripheral nerve surgery is difficult and tedious at best. Do all your nerve work by *sharp dissection* only; blunt dissection, ripping, rubbing or tearing structures in this work, once the nerve has been brought into view, is a serious mistake. By passing a curved smooth probe or buttonhook under the nerve you have full control of it.

interrupted sutures placed close together, being sure that all exposed neurons are replaced within the reconstructed sheath, because if one or more of the axis cylinders is left exposed, a "wild growth" of these cylinders develops later, resulting in a painful neuroma, or causalgia. This is particularly demonstrated in the median
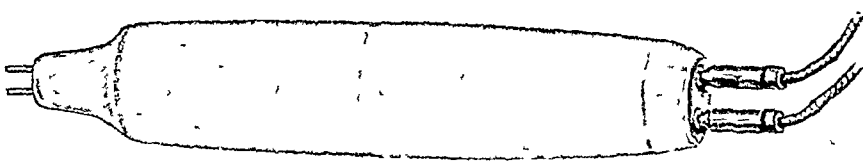
FIG. 3.

You should avoid bruising or picking up the nerve, even with blunt forceps, any more than necessary. Use a small, very sharp pointed scalpel in nerve dissection. (Fig. 1.)

In dissecting out a *freshly cut nerve*, find the severed ends, and if they are ragged, trim them off flat so that the sheath and neuraxons are clean cut and can be approximated end to end. Having prepared

nerve in the arm, and in the internal popliteal in the leg; I saw one case in the ulnar (causalgia) during my War service.

There are two schools of thought in the use of material for nerve suture; one advocates the use of very fine silk, the other favors fine catgut. Personally, I always use the catgut, for silk never absorbs but produces some fibrosis of the tissues, a

neuromatous formation, and a poor or unsatisfactory result; in using very fine tanned catgut sutures *without tension*, the

lesion, and if no muscle response occurs distal to the suspected lesion, you know the nerve is severed. (Fig. 3.) You are fully
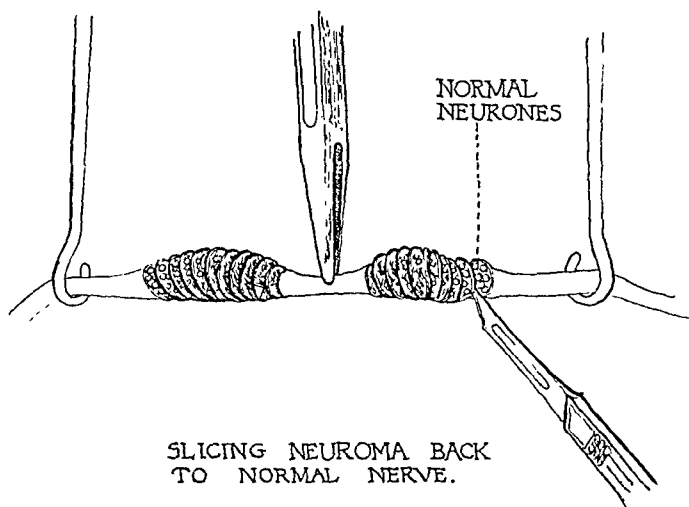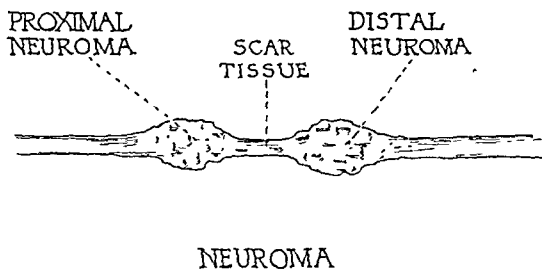
PROXIMAL
NEUROMA.　　SCAR
TISSUE

DISTAL
NEUROMA

NEUROMA

NORMAL
NEURONES

SLICING NEUROMA BACK
TO NORMAL NERVE.

FIG. 4.

catgut is absorbed in forty-five days or sooner and produces no fibrosis. This was very definitely proved by the British surgeons during the War. Since that time in civil practice I have seen a number of cases where silk was used, with failures as results.

In *delayed repair of severed nerve*, a month or more after the original trauma, you will find on exposing the nerve, a gap in the nerve substance where severed. The gap is filled with scar tissue and there is a neuromatous bulb at each end of the cut nerve, with the bulb on the proximal end usually the larger. In testing the nerve function, apply a sterilized faradic point to the nerve (under low power) above the

justified in cutting through the scar tissue in the nerve gap; then, picking up one end at a time, cut transversely across the nerve end, slicing at close intervals until you have exposed and seen the normal axis cylinders in cross-section lying like a closely packed bunch of cables in the nerve sheath. (Fig. 4.) In the operating room we use the faradic point with the tissues exposed. Therefore we cannot use ethylene anesthesia safely, and accordingly, we rely on ether anesthesia.

Of course, you must be careful in this nerve trimming process that you are not placed in the embarrassing position of making such a large "nerve gap" that you are unable to approximate the ends of the

nerve for suturing. To close the gap, as previously described, you have recourse to stretching the nerve as well as flexing

making the new bed as smooth as possible, because with a careful closure of the nerve sheath there will be no troublesome ad-
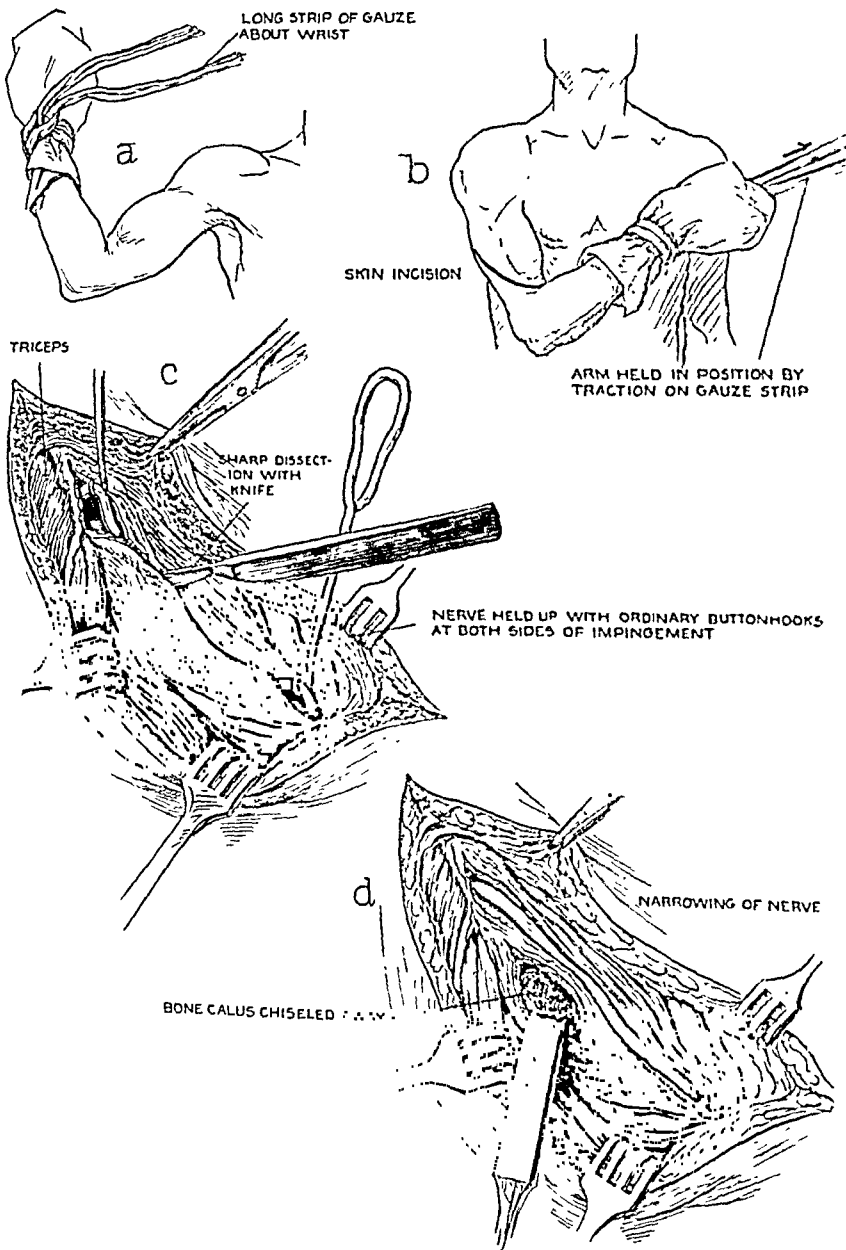
LONG STRIP OF GAUZE ABOUT WRIST

a

b

SKIN INCISION

ARM HELD IN POSITION BY TRACTION ON GAUZE STRIP

TRICEPS

c

SHARP DISSECT-ION WITH KNIFE

NERVE HELD UP WITH ORDINARY BUTTONHOOKS AT BOTH SIDES OF IMPINGEMENT

d

NARROWING OF NERVE

BONE CALUS CHISELED

FIG. 5.

the adjacent joints as you trim the nerve, in order to be sure you can suture the nerve ends together *without tension*. In delayed nerve suture there is no necessity for protecting the reconstructed nerve by placing the fat or fascia around the nerve suture; you need simply debride the tissue bed where the repaired nerve will be placed, removing all tags of tissue and

hesions. *Never* transplant a nerve *through* a muscle belly, as the muscle fibers will attach themselves to the nerve sheath and cause a constriction with a resultant causalgia or dysfunction which will require another operation and placing the nerve in a smooth bed to avoid adhesions.

Even a delayed nerve suture can lead to a good functional recovery if the work is

properly carried out, as shown in the later case reports.

Nerve suture should be done under and repair easier in a bloodless field. In the arm (above the elbow) you can safely use a tourniquet up to one hour, and above the
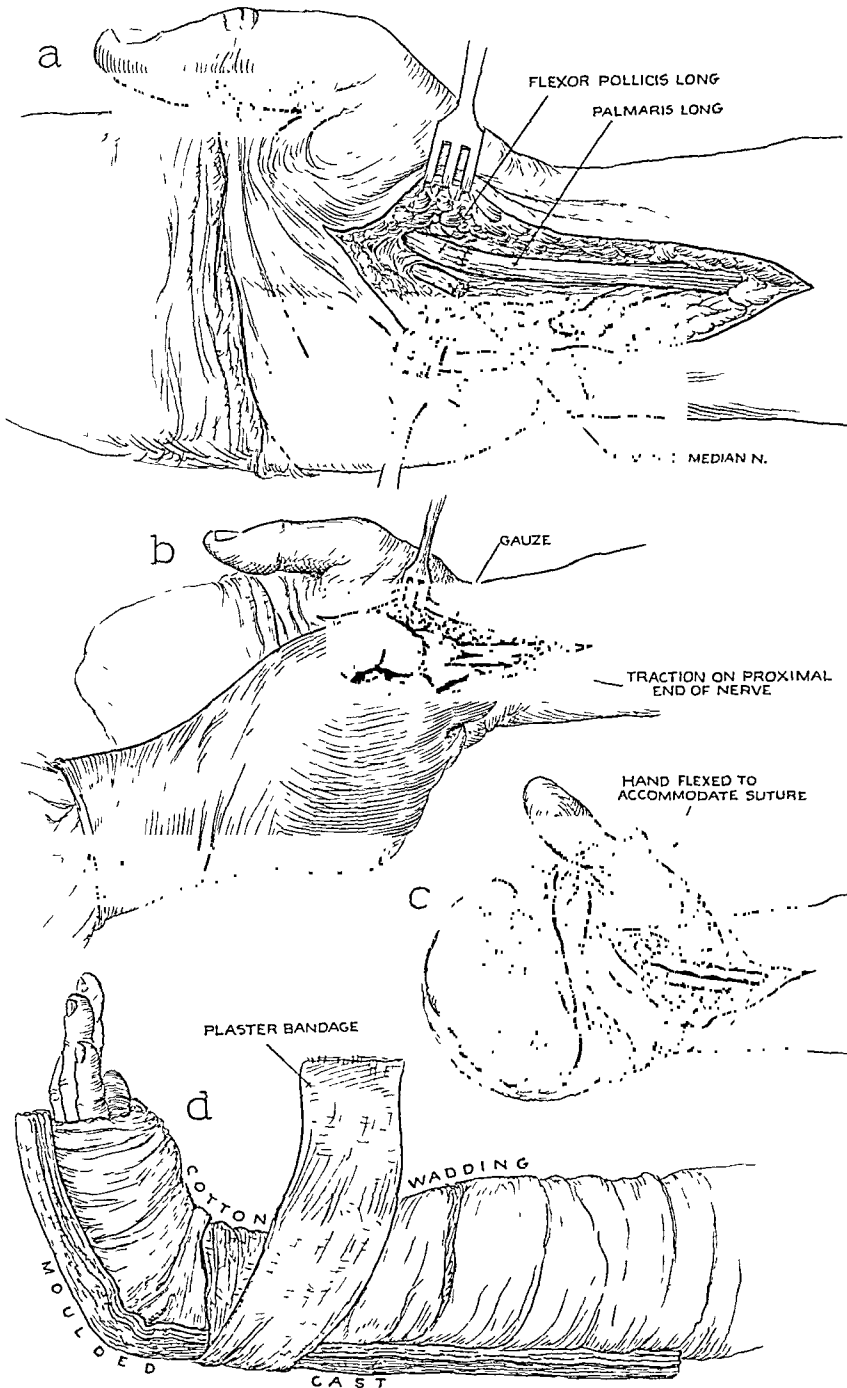

a

FLEXOR POLLICIS LONG

PALMARIS LONG

MEDIAN N.

b

GAUZE

TRACTION ON PROXIMAL END OF NERVE

HAND FLEXED TO ACCOMMODATE SUTURE

c

PLASTER BANDAGE

d

COTTON

WADDING

MOULDED

CAST

FIG. 6.

general anesthesia and it is often advisable to use a tourniquet to render the dissection

knee for two and one-half to three hours. On the arm we prefer to use a Martin

rubber bandage, for it cuts in on the soft tissue less than an Esmarch does; on the thigh an Esmarch is best.

3. *Musculocutaneous Nerve.* In my War and civil practice I never saw an injury to the musculocutaneous nerve (except in
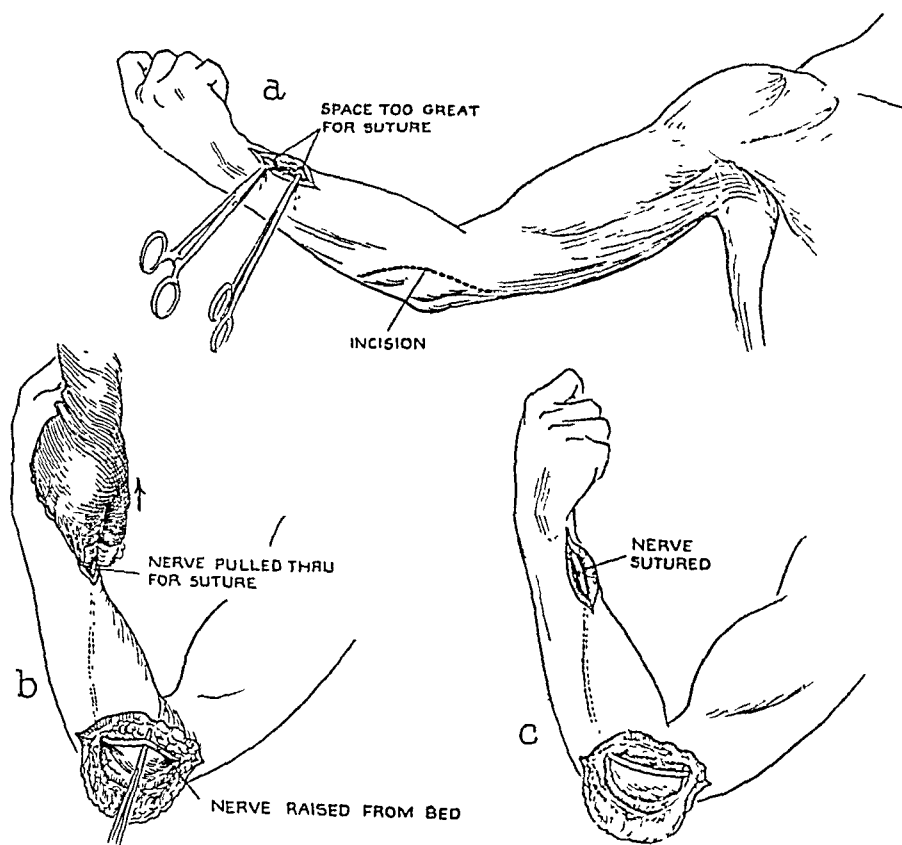
Fig. 7.

COMMENTS ON INDIVIDUAL NERVES

1. *Brachial Plexus.* Where the roots are avulsed or severed at their emergence from the spinal foramina, suture is usually impossible, or useless if attempted. Where severed in the base of the neck, suture can be done with more or less success. In the War we learned to resect a portion of the clavicle and ligate the subclavian vein in order to gain sufficient exposure to allow a successful suture of the brachial plexus. Injuries to the plexus are seldom encountered in civil practice.

2. *Circumflex Nerve.* Suture of this nerve is very difficult and as a rule not successful, due to its deep position under the shoulder muscles. If suture is attempted, the approach must be from behind the shoulder (refer to anatomy).

amputations or hopelessly destructive lesions of the arm). This nerve makes its exit out of the belly of the biceps at the junction of the lower and middle thirds of this muscle, supplying the biceps brachii, coracobrachialis anticus. Distal to that it is purely a sensory nerve, hence the rarity of any important injury to it.

4. *Musculospiral Nerve.* The usual location of a traumatic lesion of this nerve is in the arm at about the junction of the middle and lower thirds of the humerus where it is often injured in fractures at this point. The nerve may be severed, caught between bone fragments, or later incorporated in the callus with a resultant paralysis. (We have operated in two unusual cases, one a simple nail puncture, another a sliver of steel.) The surgical approach to this nerve is started at a point

10 cm. proximal to the external condyle and the incision follows a descending line from the back of the intermuscular septum
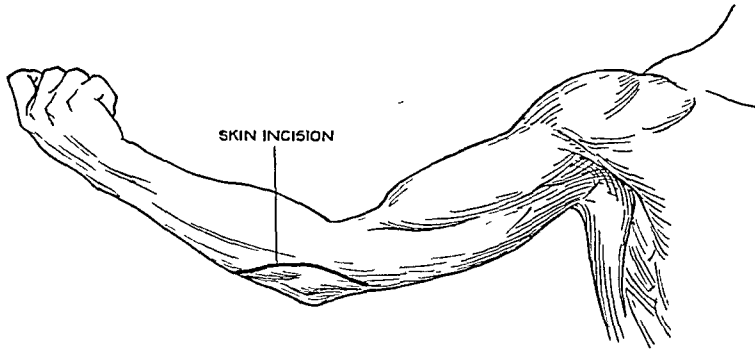

FIG. 8A.

distally and anteriorly to the antecubital space.

Like all nerve sutures, suture of the musculospiral must be done without tension. Hence the elbow should be maintained at a right angle for forty-five days after operation, preferably in a cast, with a window cut for redressing of the incision;

Fig. 8B.

during the period of recovery, the wrist drop should be corrected by a cock-up splint until voluntary muscle action has recovered sufficiently to accomplish hyperextension of the wrist and fingers. After the first forty-five days the elbow should be extended gradually for a period of two or three weeks or longer, until it can be fully straightened, thus avoiding any abrupt stretching of the healing nerve. On cast removal, use sinusoidal galvanism treatments three times a week for six to eight

months if necessary, over the affected muscles, to maintain muscle tone and volume until such time as the nerve regenerates and functions; even after that you should continue sinusoidal treatment until you are satisfied that you have gained all you can in recovery.

In immediate suture of the musculospiral the results are good, with sensory function appearing in the web between the thumb and index finger dorsally in about
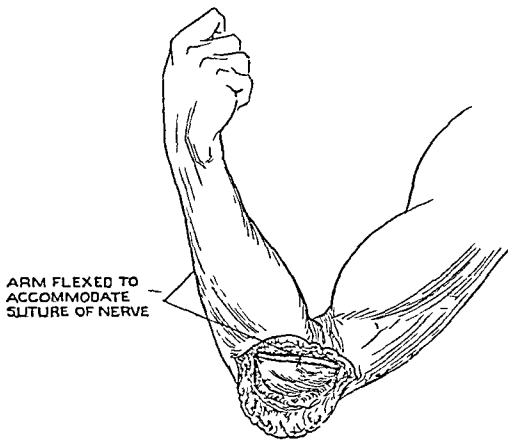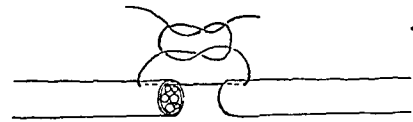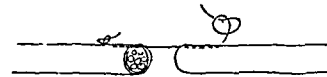

SUTURE OF
NERVE IN HAND

SUTURE OF SMALL
NERVE IN HAND
FIG. 9.

two or three months after nerve suture, and voluntary muscle response (extension of wrist and digits) in about four to six months. Faradic response usually shows in about six to eight months and improvement continues for a year, occasionally longer, after suture. Length of temporary total disability depends on the patient's occupation, of course. The functional cure

is complete when the patient is able to place the dorsal surface of his forearm and hand on a table and extend all the fingers until he touches the tips to the table.

elbow completely, suturing the nerve end to end in that position. We found we could close a gap of 2 inches (maximum) by wrist flexion, combined with elbow flexion.
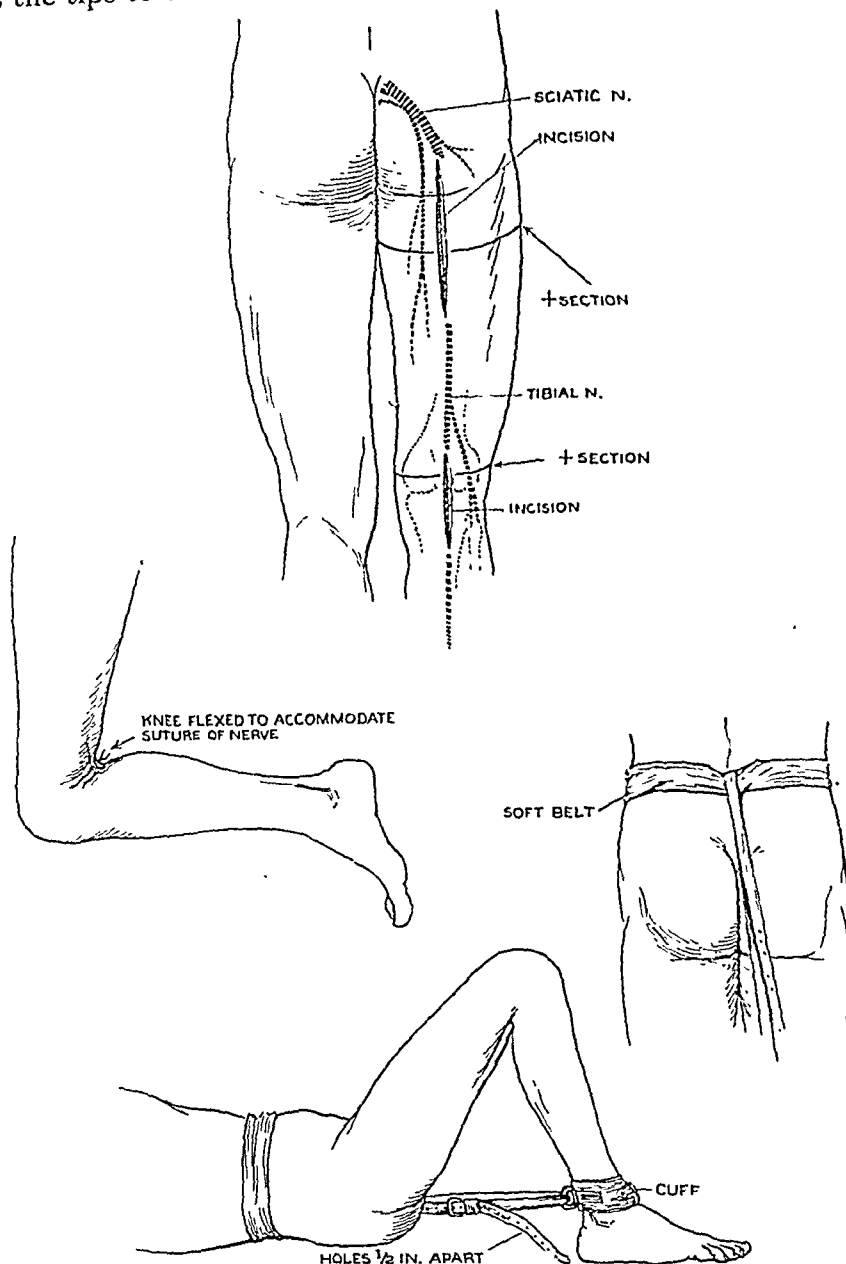

Fig. 10.

5. *Median Nerve.* This is most frequently injured at and just proximal to the wrist level, as seen in civil practice, although I have seen it injured at or near the elbow in the War, due to gunshot wounds.

In the War injuries we found we could bridge a gap in the median nerve at the wrist or elbow by stretching the proximal end of this nerve and flexing the wrist or

This position is maintained during nerve suture and after closure of the tissues in a cast for forty-five days. Then the wrist and elbow are extended about an inch at a time until the member is brought back to normal. After cast removal, sinusoidal galvanism is given about three times weekly, as described under the musculo-spiral nerve technique.

It is surprising how small a wound at the wrist can involve the median nerve; anatomically, it lies alongside of and
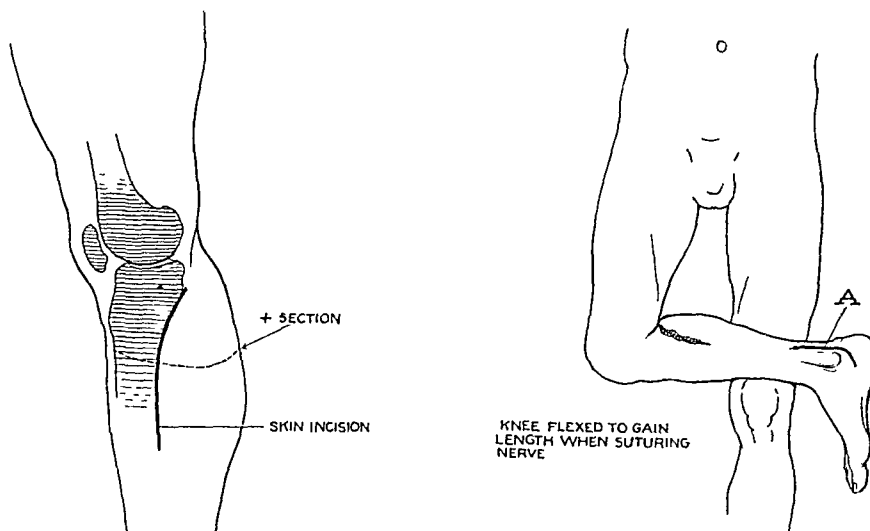

FIG. 11.

slightly deeper than the palmaris longus tendon, so the nerve can be injured easily in wrist wounds. Whenever you are faced with a volar wound at the wrist, a careful test *and exploration* should be made for both median and ulnar nerve injuries. These nerves are very important in the function of the hand, and the sooner a nerve repair is made the better the end results will be. If six hours or more have elapsed since injury occurred, you had better delay nerve suture for fear of infection, unless you feel you can thoroughly debride the tissues. Otherwise, wait a period of about two to three weeks in dealing with a cleanly healing wound; or two to three months after infection subsides, if infection does occur. A wound infection is disastrous, of course, in its effect on a nerve suture, when repair is attempted under such circumstances.

A return of nerve sensation usually can be detected in an average time of two to three months after the immediate suture, and progresses to complete or almost complete sensibility. The return of the motor function is slower, usually being evident in five to six months and progressively improving. Voluntary muscle control, then

Faradic response appears. Sinusoidal galvanism, massage and exercises should be continued for six to eight months at least, and usually longer if the patient will permit. The maximum of recovery usually takes place in one to one and one-half years. Disability depends on the patient's occupation.

Median nerve causalgia is an occasional result of injury to this nerve when untreated or improperly sutured, and is very painful and disabling.

6. *Ulnar Nerve.* This is severed or injured most commonly at the wrist level, and next most commonly at the elbow. At the wrist it lies superficially enough, close to the ulnar vessels and flexor carpi ulnaris tendon so that it is injured just as easily as the median nerve. In the repairing of the flexor carpi ulnaris or the stopping of hemorrhage from the ulnar vessels, the ulnar nerve severance is frequently overlooked by a careless exploration, and a late repair is necessitated. The ulnar nerve paralysis is very crippling to the hand, and fingers, not only in the numbness resulting but in the muscle effects, hence the importance of immediate repair which yields so much better a result than delayed nerve suture.

The technique (Figs. 7 and 8) is the same as in median nerve suture at the wrist,

except that if a large nerve gap has to be closed, one must gain length of nerve by flexing the wrist completely and, if neces-

return of motor function is more uncertain and incomplete. The earlier the nerve suture can be done, the more complete is

FIG. 12.

sary, making a second incision at the medial aspect of the elbow and transplanting the nerve forward of the condyle, then flexing the elbow; a considerable gap (at least 2 inches) in the nerve at the wrist can be closed by traction on the proximal nerve end, and by right angle flexion of the wrist and elbow. The after treatment is the same as described for the musculospiral and median nerves (forty-five days in cast, followed by massage and sinusoidal galvanism three times a week, etc.). Sensation begins to return in two to four months in successful suture, and motor voluntary functions begin to show in four to six months as a rule. Recovery is slow in the ulnar, taking one and one-half to two years, during which time medical treatment (such as sinusoidal galvanism and massage) and observation preferably should be continued.

The more proximal ulnar lesion, at the elbow, recovers more slowly after nerve suture, as the neuraxons have so much further to regenerate. A fairly good return of sensory function can be expected, but

the eventual recovery, as muscle atrophy and fibrosis and contracture of the ring and little fingers do not have a chance to become apparent.

7. *Nerves of the Hand and Fingers.* In the hand they can be sutured with success if carefully done, though this is very difficult work. On the branches of the median and ulnar nerves, a triple zero Pyoktannin catgut strand can be run through the sheath of the proximal nerve end and the distal nerve end, drawing the ends together and holding in approximation by a knot close to the sheath at each end of the suture. We have done this successfully on the median nerve branch to the middle finger in one case, and to the thumb in another case, and these patients recovered skin sensation over that nerve distribution.

The nerves of the fingers (the digital branches of the median and ulnar nerves) are so small that suturing them is out of the question. They are large enough so that a painful neuroma in the scar can be found and dissected out quite readily, using a rubber band tourniquet to render the field

of dissection bloodless. One can resect completely such a finger neuroma without attempt at nerve suture, and still leave good function in the finger.

8. *Sciatic Nerve.* This is one of the most commonly injured structures in the lower extremity, between the hip and the thigh. Injury to the sciatic nerve is seen only occasionally in civil practice, although it was seen quite commonly during the World War in my experience. We found that any attempt to suture a sciatic lesion proximal to the gluteal fold was impractical, because anatomically this nerve begins to bifurcate at the lower border of the quadratus femoris muscle and interlacing this area is a vast arterial supply which surrounds the nerve at this junction. Attempting nerve suture here would result in extensive hemorrhage and possible death. The reader can study out the detailed anatomy of this region if he desires.

Between the gluteal fold and the popliteal space this nerve can be sutured, and can be brought together where there is a gap of as much as 5 inches. This is accomplished first by making a medial incision, exposing both nerve ends, using strong traction on the distal portion of the nerve and thus gaining a stretch of 2 inches (and there is no harm done in stretching gently the proximal end); then the knee can be flexed to a right angle or more, thus giving the operator another 3 inches of nerve. The ends can be approximated and sutured without tension in this position, the limb maintained thus for at least forty-five days by the use of a belt around the abdomen, another round the ankle, and running between the belts a leather strap and buckle with belt holes one-quarter inch apart. At the end of forty-five days, you can begin to let the knee straighten and the leg come down, one hole at a time, and you will find that the natural weight of the lower extremity will stretch the nerve gradually without tearing the sheath sutures apart. Meanwhile sinusoidal galvanism can be used on the muscles below the seat of the lesion, the treatment being rendered at least three

times a week. This will maintain the muscle tonicity and volume until such time as the nerve has regenerated sufficiently to resume its responsibilities. I wish to emphasize again that sinusoidal galvanism is used (in these treatments after nerve suturing) and not Faradism (the latter is useful only in diagnosis).

Prognosis: A successful suture requires one and one-half to two years for recovery. Disability runs nine to twelve months. There is always a temporary residual foot drop in these cases, which can be controlled by the use of a prosthetic shoe holding the foot at right angles while walking.

9. *Internal Popliteal Nerve.* Keep in mind that some 4 inches above the popliteal space the sciatic makes its division into the external and the internal popliteal nerves, the internal passing down through the popliteal space under the inner head of the gastrocnemius to perforate the soleus muscle and lie to the inner side of the leg, passing down to the ankle where it bifurcates again into the two plantar branches.

My reason for going into this detail is that if a lesion of this nerve exists below the popliteal space you must not try to reach the nerve (at operation) by placing the patient on his abdomen and going between the two bellies of the gastrocnemius; but have the patient placed on his back, cross the affected limb over the other (Fig. 11) and make incision anterior to the gastrocnemius, drawing the belly of the inner head to one side, thus exposing the soleus muscle; pass through the fibers of the soleus and you can reach the internal popliteal nerve easily. A gap of at least 2 inches in this nerve can be closed by flexing the knee, the same as one would do in a high sciatic.

Prognosis: One to two years for full recovery. Disability endures for five to six months, and then a prosthetic shoe holding the foot at right angles should be worn until full recovery.

10. *External Popliteal and Peroneal Nerves.* Keep in mind that the anterior

tibial nerve passes around the outer side of the head of the fibula, and as it does so, it breaks up into branches to supply the

Fig. 13. Case 1.

extensor muscle group, the peroneal division supplying the peroneus longus and brevis. It separates from the external popliteal in the popliteal space. It is therefore always advisable to explore these branches where there is a wound in the popliteal space.

Prognosis: One to two years for recovery. Disability five to six months with a prosthetic drop foot shoe.

11. *Anterior Crural Nerve.* This nerve I have seldom found to be injured in my experience in civil practice, and I saw only two cases of this lesion during my War experience; in fact, the only region in which this nerve can be affected is the groin or just distal to it. A wound in this location is almost certain to involve also the closely associated femoral artery and vein, and usually death results from hemorrhage or

gangrene of the leg. In the two War cases above mentioned, curiously enough, through some unusual direction of the striking force, the anterior crural nerve alone was involved and both patients lived. In civil practice, we have seen one case where an ice pick penetrated the femoral region and severed the nerve and vessels, resulting in gangrene and amputation. Prognosis is one to two years for recovery. Disability one to two years.

CASE REPORTS

Following are brief descriptions of some actual cases in which we have performed early or late nerve suture, illustrating the technique, after-care, progress and results in the more common peripheral nerve lesions:

CASE 1. Suture of the musculospiral nerve above elbow (two months after injury). End result at thirteen months.

C. I., age 35, a married truck washer, was injured June 8, 1937. A truck tire rim shattered and caused a small puncture wound above the

Disability was prolonged due to a delayed union in a compound fracture of the right leg caused in the same accident, and the fact that

FIG. 14. Case VI.

right elbow; the wound was repaired elsewhere and was seen by us eight hours after injury. Complete musculospiral paralysis was present.

We used a cock-up splint for the wrist, waited until August 14, then we operated, found the nerve severed 2 inches above the elbow, scarred and bulbous, with an encysted steel fragment near it. The nerve was sutured, the arm kept in a cast with elbow flexed until forty-five days postoperative, then the elbow was gradually extended. Sinusoidal galvanism, heat lamp and massage once weekly were started September 28, 1937. By October 22, 1937, there was weak response in the wrist and finger extension to sinusoidal stimulation, but no voluntary muscle action. By November 29, a flicker of voluntary extension appeared at the wrist. Hyperesthesia was present over the thumb web dorsally by January 26, 1938. By March 22, 1938 he could extend the wrist 50 degrees. Sinusoidal galvanism and massage were continued until March 31, 1938 (eight months postoperative).

the patient found difficulty in obtaining a job. He actually returned to work July 1, 1938, about thirteen months after injury, as a W. P. A. laborer, also working on his free days as a washing, greasing and gasoline attendant in a garage, and continued this work. Our last examination, September 7, 1938, showed full motion of elbow, wrist (70 degrees extension) and fingers, slight impairment in strength of thumb extension (though full range), slightly impaired and hyperesthetic sensation dorsally at thumb and thenar web. He thinks he is still improving in motion.

CASE II. Delayed lysis (seventeen days) of pinched musculospiral nerve.

L. R., age 18, single, schoolgirl, was injured May 10, 1936 and was first seen by us eleven days after injury. She had sustained a transverse fracture of the left humerus at the junction of the middle and lower thirds, with complete musculospiral nerve paralysis, total wrist drop, and fracture displaced. At operation on May 26, 1936, seventeen days after

injury, we found the nerve caught and constricted between bone fragments; it was freed, the fracture reduced with an intramedullary

aspect of the right wrist, with median nerve severance. He was first seen by us seven months after injury and operated on December 17,

FIG. 15. Case VII.

beef bone peg, and the nerve transplanted to extramuscular position. A cast with elbow flexed was applied. Two and one-half months postoperatively, we started sinusoidal galvanism twice weekly, with heat and massage. Three and one-quarter months postoperative we removed the posterior mould when bone union was firm.

On September 10, 1936, nerve function was returning and the patient could extend the wrist a few degrees; by December 15, 1936, full nerve recovery was evident (seven months postoperative). After another month the patient was discharged from treatment with elbow extension restricted 35 degrees. End result at one and one-half years postoperative: full nerve recovery, elbow extension restricted 20 degrees.

CASE III. Late repair (seven months) of median nerve at wrist. End result at one year and ten months.

J. H., age 22, single, barman, was injured on May 20, 1935 by a glass cut on the volar

1935. We found a completely severed median nerve, just above annular ligament, with a pseudoneuroma on the proximal end of the nerve. The neuroma was cut back and sutured, flexing the wrist. A cast was in place for forty-five days.

The patient was discharged from the hospital in three days. Six weeks postoperative sinusoidal galvanism was begun, with gradual extension of the wrist. Massage was started eight weeks after operation. In five weeks the patient showed some sensory regeneration to the index and middle fingers and the end of the thumb. He was discharged from treatment August 5, 1936 (seven and one-half months postoperative), after he had resumed work as a barman the month before.

Examination one year postoperative showed full range of digital motion with no muscle atrophy, but the patient claimed some impairment in sensation over the median nerve distribution to the hand and digits.

CASE IV. Late repair (thirteen months) of ulnar nerve at elbow.

Mrs. J. E. M., age 35, housewife, was injured

A. H., age 42, married, with two children, a milk handler, was injured on July 19, 1935, when he fell from a ladder and cut his left
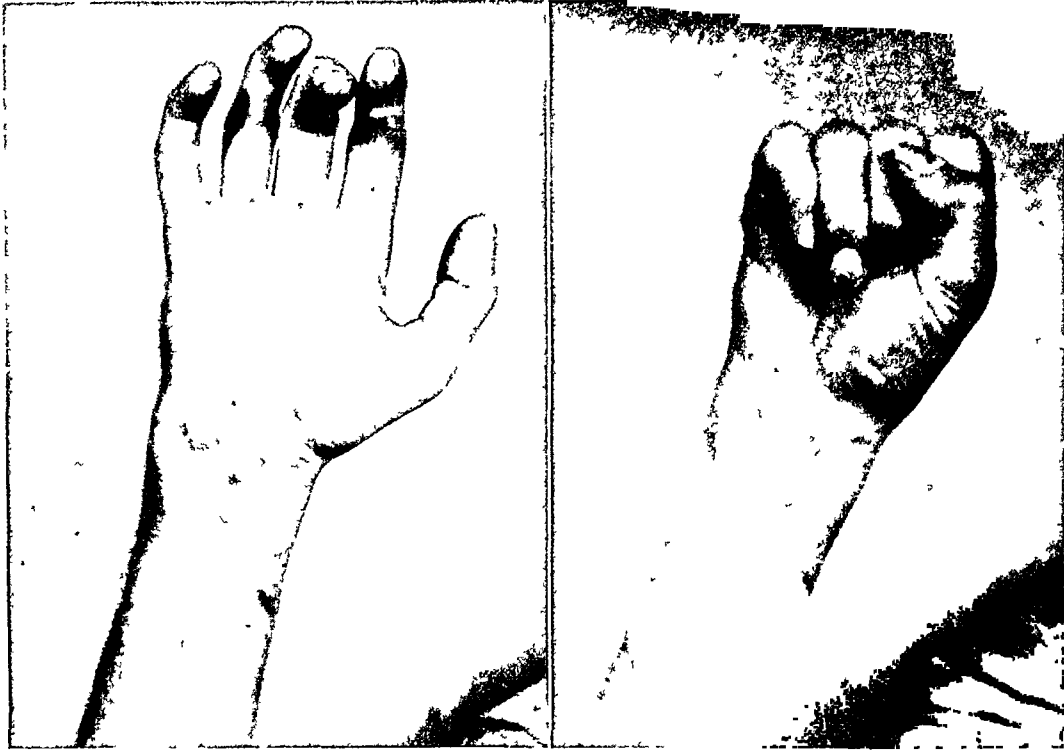

FIG. 16. Case VIII.

thirteen months prior to our first examination. She suffered complete ulnar nerve paralysis and extensive scars at the medial and posterior aspects of the elbow and complete flexion contracture of the ring and little fingers. At operation by us July 22, 1937, the ulnar nerve was found severed at the ulnar groove and its ends separated 3 inches, with a neuroma of the proximal end of the nerve. The neuroma was resected, the ends of the nerve sutured, with the wrist and elbow flexed to close the gap. A cast was worn for forty-seven days.

On October 22, 1937 (three months postoperative) there was partial recovery of sensation in the hand and fingers mingled with areas of hyperesthesia. The patient could voluntarily extend the ring and little fingers 20 degrees. Further recovery was expected, but the patient has been unable to come to us for reëxamination as she lives in a distant city. We have heard, however, that she has further improved in sensation and motion.

CASE V. Delayed repair (seven weeks) of ulnar nerve at elbow. Later nerve transplant. End result at two years and one month.

elbow. Some wound infection followed, the wound was sutured by a local doctor. We first examined the patient five weeks after injury and found complete ulnar paralysis distal to the elbow with beginning muscle atrophy, anesthesia and trophic changes.

Operation was performed September 11, 1935, seven weeks after the injury. We found the ulnar nerve severed at the elbow and neuroma formation. The nerve was sutured and passed through the intramuscular tunnel in front of the internal condyle with the elbow flexed. A cast was applied.

Three days after operation the patient left the hospital. The cast was removed forty-two days postoperative, and sinusoidal galvanism was started. The elbow was gradually extended, and sensation was recovered down to the wrist. Treatments were given twice weekly.

The patient returned to work December 9, 1935, three months postoperative. Five months from date of operation we found beginning ulnar causalgia with a painful palpable lump at the point of suture (adhesions). On August 4, 1936 (ten months after the first operation) a

second operation was done, dissecting the scar, freeing perineural adhesions and transplanting the nerve outside the muscles. Causalgia ceased at once.

By October 5, 1936, thirteen months post-operative, there was almost full return of sensation in the hand and fingers, although the little finger remained contracted 25 degrees. The patient used his left hand dumping 40,000 pounds of milk a day from eight-gallon cans. There was an estimated 15 to 20 per cent specific loss in the left hand.

End result on October 26, 1937, two years and one month postoperative, showed almost full sensory recovery and only slight thenar web and interosseous muscle atrophy. The little finger was curled in 45 degree flexion.

CASE VI. Immediate suture of ulnar nerve at wrist, and four flexor tendons. End result at three and one-half years.

E. W., age 20, single, a porter, was cut by glass May 17, 1934. Operation was done immediately by us, consuming two and one-half hours. The ulnar nerve and its dorsal cutaneous branch, which were severed, were sutured, and four cut flexor tendons as well. The median nerve was exposed and found to be intact. A cast was applied with wrist flexed.

The patient was discharged from hospital in seven days, but the cast maintained for forty-five days. Then sinusoidal galvanism, heat and massage were started, three times weekly. Ulnar sensory return was first noticed two and one-half months after injury. The patient was discharged from treatment six months post-operative. December 5, 1934, the compensation claim was settled before the Industrial Commission for 20 per cent functional loss in right hand.

The end result at examination on October 16, 1937, three and one-half years after injury showed full ulnar nerve recovery except for slight atrophy of the thenar web and slight restriction in adduction and extension of the ring and little fingers. Full flexor tendon recovery had occurred in the four fingers.

CASE VII. Late suture (two and three-quarter years) of ulnar nerve at wrist. End result two years and ten months after operation.

S. V. B., a schoolgirl, age 13, was injured about two years and nine months prior to our first examination on January 3, 1935. A glass cut of the left wrist had left complete ulnar paralysis distal to the scar, with complete contracture of the ring and little fingers. Operation by us on January 5, 1935 showed the ulnar nerve completely severed just proximal to the wrist. The nerve damage was resected and the 1½-inch gap closed by stretching the nerve, flexing the wrist, and suturing the nerve. A cast was worn for fifty-one days, with wrist and elbow flexed.

Sensory recovery was evident beginning thirty-seven days postoperative. Eight weeks postoperative sinusoidal galvanism, heat and massage were started, twice weekly. Three and one-half months postoperative, voluntary motor response was noted in fingers. Six months post-operative, full sensory restoration was present except for dulness to touch and pain at the tip of the little finger. Treatments were then resumed for eleven weeks.

End results two years and ten months after operation: Full sensory return (touch, pain and temperature) in left hand and fingers, but only about 20 per cent recovery of muscle volume and control; some permanent fibrous degeneration and muscle atrophy were noted.

CASE VIII. Delayed suture of median and ulnar nerves at wrist (seven weeks) and tendon repairs. End result at three years four months.

Mr. R. Z., age 25, married, a truck driver, was injured May 7, 1934, when he fell, cutting his wrist on brass objects. First aid was rendered elsewhere with supposed repair of the flexor tendons. Our first examination three days after injury, showed ulnar and median nerve paralysis distal to the wound, and flexor tendon loss of function to all fingers.

Our operation was done June 29, 1934, seven weeks after injury. The superficial flexor tendons of the middle, ring and little fingers were found sutured to the proximal end of the ulnar nerve, leaving the distal end of the nerve free; median nerve found sutured to superficial flexor tendons of index and middle fingers, distal end of nerve free. There was marked interstitial fibrosis of the median nerve. All structures were repaired, flexing the wrist to close the nerve gap; the flexor carpi ulnaris tendon was transplanted to the superficial flexors of the middle, ring and little fingers because shrinkage of the severed tendons had made their junction impossible. A cast held the elbow and wrist flexed 90 degrees.

The patient was discharged from the hospital in six days. The cast was bivalved in one month and sinusoidal galvanism started, three times

weekly. The cast was removed seven weeks postoperative.

On November 24, 1934, a Volkmann splint was applied to stretch the flexor tendons. At the end of six months ulnar sensation was returning with hyperesthesia. Eleven months after operation median nerve sensation was present in hand and fingers, also ulnar sensation with decreasing hyperesthesia. Thirteen months postoperative the patient returned to work. A month later he was discharged from treatment. Fifteen and one-half months postoperative the claim was settled before the Industrial Commission for 50 per cent specific loss of right hand.

End result three years four months postoperative; full return of median and ulnar nerve function, both sensory and motor, 20 degrees flexion contracture of index, ring and little fingers and 50 degrees contracture of middle finger. 5½ inch operative scar. Some bulging was present in the region of the annular ligament.

CASE IX. Late suture (four and one-half months) of sciatic nerve at knee. End result seven and one-half years.

G. V. was injured October, 1930, by a bullet wound behind the left knee. The external and internal popliteal nerves were completely paralyzed. On March 16, 1931, nerve suture was performed by us, five and one-half months after injury. Suture was done under local anesthesia (general anesthesia was contraindicated by a kidney complication).

The peroneal tendons began functioning five and one-half months postoperative. The patient returned to work nine months after operation (he was willing to return to work sooner but his attorney forbade it due to his claim pending settlement).

Examination by us on June 24, 1938, seven and one-half years postoperative, showed: circumference of right calf 14¼ inches; left calf 13¼ inches. The anterior tibial muscles did not

function completely, but some anteflexion of foot was produced through the peronei; no Faradic response was present in the anterior tibial group, but strong voluntary action and Faradic response were evolved in the peronei and the posterior group of muscles; the external popliteal nerve suture had resulted very successfully. Sensory return over the anterior tibial distribution was complete (sensation present in the triangle between the great and second toes).

SUMMARY AND CONCLUSION

Peripheral nerve injuries are discussed in general considerations, diagnosis, detailed technique of nerve suture operations; the after-care, prognosis and results. The more common nerve injuries are described individually, and examples are given in nine case reports of early and delayed nerve suture. A plea is made for early and correct diagnosis, and proper suture and after-care of these important nerve injuries. You can see by these case reports that one can be mistaken very easily in operating on these cases in suturing a tendon to a nerve; but if proper time is taken to study carefully the associated structures, the operator cannot help but see the difference, in that a nerve contains cables of nerve structure where the tendon has a flat surface over cross section. It is very important to study these structures and be sure that you are suturing nerve to nerve and tendon to tendon. These mistakes often cause disability that cannot later be repaired completely.

PATHOLOGIC FACTORS FOUND IN THE SURGICAL INVESTIGATION OF EPILEPSY

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THE chronic convulsive state commonly known as epilepsy has so long been considered by neurologists as a cerebral agenesis or degeneration with hereditary tendencies that an attempt to present causative pathologic factors is apt to be received with considerable skepticism, and particularly so when considered surgically. Neurologists do, however, suspect a local cerebral lesion and occasionally invite surgical exploration when attacks are jacksonian in character, or when generalized convulsions are preceded by a definite localizing aura. In the absence of clinical signs of a well-localized cortical irritation, surgical investigation of the epileptic brain has been generally discouraged. This attitude is based on the failure of surgeons to demonstrate a definite pathologic condition which could reasonably be accepted as a causative factor, and on the fact that most surgical procedures have failed to influence the epileptic reaction favorably.

An expanding lesion, or the scar of a localized trauma, may or may not produce sufficient cortical disturbance to induce convulsive phenomena, but, when their occurrence precedes the incidence of attacks, they are usually considered causative, particularly so when attacks exhibit focal symptoms. The great majority of patients presenting clinical phenomena characteristic of epilepsy exhibit no definite focal symptoms. Focal attacks are of great importance from the standpoint of localization when substantiated by neurologic findings but their absence is not sufficient to eliminate even a well-localized lesion, for many brain tumors do not instigate convulsive seizures with jacksonian features, and this is also often true of attacks following local cerebral traumatic lesions.

The "idiopathic" type of epilepsy comprises that great group having periodic epileptic reactions without neurologic evidence of a local cortical irritation, an increase of intracranial pressure, or a definite cerebral traumatic history. They usually present no abnormality demonstrable clinically other than the epileptic reaction, and most patients are apparently perfectly normal between attacks, except that when seizures become frequent and prolonged they usually show signs of mental deterioration. The occurrence of convulsive phenomena (particularly group attacks) in patients who are apparently normal between attacks suggests that there must be some periodic factor active which has escaped recognition. The gliosis so commonly found in the post-mortem examinations of institutional epileptics, i.e., advanced cases, can be attributed to the vascular changes produced by the severe cerebral congestion attending convulsive attacks. There is much evidence to support the assumption that such changes are the result, rather than the cause, of convulsive phenomena.

Convulsive phenomena often attend certain expanding lesions where there is an increase of intracranial pressure; they also occur after cerebral trauma in which there has been a loss of brain substance resulting in an intracranial hypotension. Convulsive phenomena are not dependent on, or even a characteristic reaction of, a general increase of intracranial pressure. Convulsive attacks may develop with the local expansion of a brain tumor, cease after tumor removal, and later recur without a recurrence of the neoplasm. Clinically, the epileptic reactions associated with brain tumor or cerebral trauma do not differ from those seen in the so-called "idiopathic" type of

epilepsy. Even when they are jacksonian in nature, the convulsive reactions of an expanding lesion do not differ from those of a traumatic lesion, and the differentiation between such lesions must be made on grounds other than the observation of attacks.

Thus, we have epileptic reactions which are clinically identical accompanying very diverse pathologic cerebral conditions, such as brain tumor and craniocerebral trauma, in which these lesions may be considered definitely causative. Evidently there exists in these different pathologic states some factor common to both which is capable of producing cortical irritation of sufficient intensity to establish ultimately an epileptogenous zone and the epileptic reaction. This common factor seems to be an increase of local cortical tension. This tension develops in the area surrounding an expanding cortical tumor, in the contraction of a scar, or the tugging on corticodural attachments in traumatic or inflammatory lesions. At operation, particularly under light anesthesia, the traction occurring during the separation of traumatic corticodural adhesions or in the finger enucleation of a tumor will commonly induce convulsive phenomena. This traction element can also be definitely demonstrated in the so-called "idiopathic" type of epilepsy, as we shall see later, where traction may be induced artificially and be followed by typical epileptic reactions. When the traction element is diminished or eliminated, there is a reduction or a complete cessation of epileptic phenomena.

The factors involved in this element of traction in the "idiopathic" type of epilepsy have to do with cerebral postural instability—a disturbance of those factors which normally maintain the brain in postural stability within the cranial box. Through a tear in the arachnoid, fluid leaks into the subdural space; the brain, losing support, gravitates to dependent portions of the skull; traction is exerted on corticodural attachments at the extreme vertex of the brain where the cortical motor centers

are located. In time, this traction produces a fairly well-localized area of cortical hyperirritability, an epileptogenous zone, which characterizes lesions responsible for epilepsy and which reacts to stimulation or traction with a convulsion.

In the roentgenologic examination of epileptics, encephalography is commonly used in the hope that visualization of the cerebral ventricles and subarachnoid space may reveal some abnormality. When air is injected into the spinal canal after the removal of cerebrospinal fluid, it ascends into the cerebral ventricles and subarachnoid space. With the introduction of encephalography, it was found that air occasionally escaped into the cerebral subdural space. Investigators of the cerebrospinal fluid pathways—Key and Retzius, Quincke, Weed,[1] and others—have concluded that normally there is no communication between the subarachnoid and subdural space which would allow fluid to escape from the former into the latter. If, after encephalography, air escapes from the subarachnoid into the subdural space, it can only do so through a defect in the arachnoid. If air escapes from the subarachnoid into the subdural space after encephalography, then it is reasonable to expect cerebrospinal fluid to do likewise. I had so commonly encountered great collections of subdural fluid when operating on epileptics for some suspected cortical lesion, that the demonstration of air in the subdural space after encephalography caused me to postulate, some eleven years ago, arachnoidal defects or fistulas through which cerebrospinal fluid entered the subdural space. The exact nature of these fistulas and their location I have recognized only during the past few years, though undoubtedly I had exposed them on many occasions.

Arachnoidal Fistulas. Arachnoidal fistulas are defects in the arachnoid through which cerebrospinal fluid leaks into the subdural space. They have thickened edges, and are usually located at the extreme vertex of the brain adjacent to the angle of
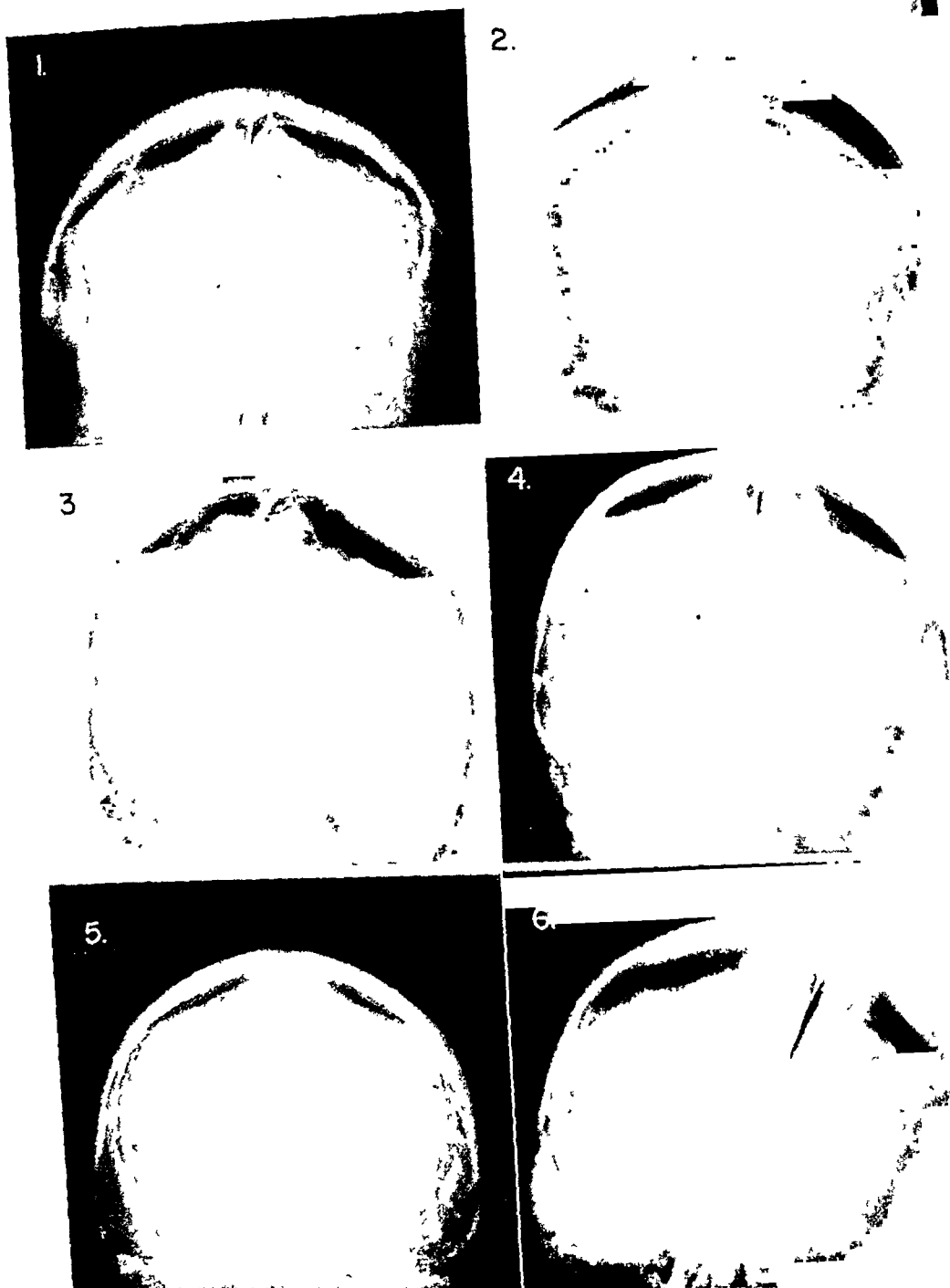
Fig. 1. A series of subdural air insufflations in cases of the so-called "idiopathic" type of epilepsy. 1, shows the slight extent of corticodural attachments adjacent to the sagittal sinus in the frontal region. 2, same patient as 1, showing dense corticodural fixations at the extreme vertex of the skull near the motor area. 3, subdural fluid level demonstrated with floating lipiodol on one side and air, as the contrasting medium, on the other side. 4, extensive corticodural fixations just anterior to the motor area in a child with rather extensive erosion of the skull. The subdural fluid level is shown by contrast with the superimposed air. 5, extensive corticodural fixations just anterior to the motor region with erosion of the skull. Lipiodol used on one side to demonstrate the subdural fluid level. 6, extensive corticodural fixations near the motor area at the vertex. Subdural fluid level demonstrated by a small amount of lipiodol on one side. Lipiodol is no longer used as a contrasting medium for subdural fluid, the superimposed air being sufficient.

a cortical vein as it turns to parallel the sagittal sinus. Since the characteristics of
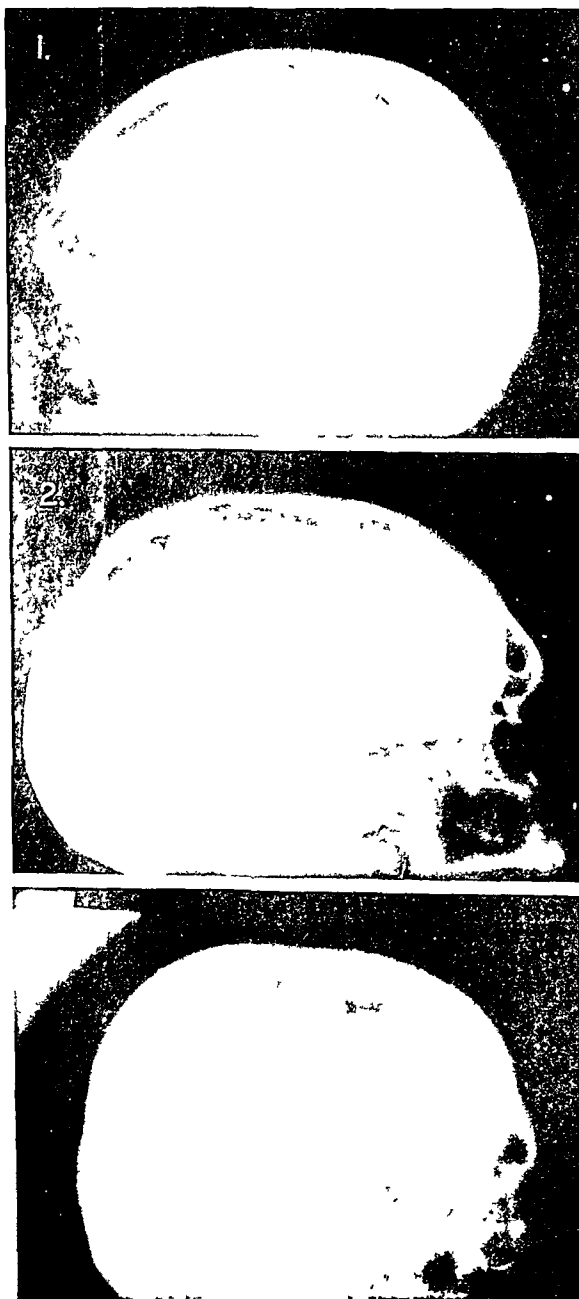

FIG. 2. Subdural air insufflation in typical cases of epilepsy. Lateral views. 1, typical corticodural fixations just posterior to the bregma involving the motor and premotor areas, with some erosion of the skull. 2, corticodural fixations in the motor and premotor areas. Lipiodol floating on the fluid shows clearly a typical fluid level. 3, subdural fluid level contrasted only with air. Corticodural fixations are shown in the motor area. In each of these there will be observed a certain amount of erosion of the bone where corticodural fixations occur.

arachnoidal fistulas have been recognized, I have found them at operation in every case of epilepsy. In exposures of the cerebral vertex, when the dura is carefully reflected toward the sagittal sinus, a constant escape of cerebrospinal fluid through these arachnoidal defects is observed. If air is locally injected into the subarachnoid space, it may be directed toward the fistula through which it will escape as bubbles. Apparently these defects are the result of trauma—small tears in the arachnoid, where it is attached to the dura along the sagital sinus, which failed to heal and have persisted as chronic fistulas. Their usual location, at the extreme vertex of the brain where the strain of gravity is greatest in the erect posture, is suggestive as to their origin and persistence.

Cerebral Postural Stability. Considering the great weight of the brain, what maintains it in a state of postural stability within the cranial box? The arachnoid is attached at the vertex along the sagittal sinus where the cortical veins turn to parallel the walls of the sinus. There is normally no organic attachment between arachnoid and dura over the lateral surface of the cerebral hemispheres. It has been generally considered that the arachnoid, distended by cerebrospinal fluid under pressure, served to hold the brain in a state of postural stability within the cranial cavity. The arachnoid over the entire surface of the brain sends numerous trabeculae to fuse with the pia, and these hold the brain nicely suspended in the center of the cranial box provided the arachnoid is held in close approximation to the dura. On this arachnoidodural approximation depends the postural stability of the brain within its container. Evidently there are factors other than the pressure of cerebrospinal fluid which maintain arachnoidodural approximation, as it is not disturbed by the withdrawal of cerebrospinal fluid even to the point of producing a negative pressure. Roentgenologic examination after a large quantity of fluid has been withdrawn and partially
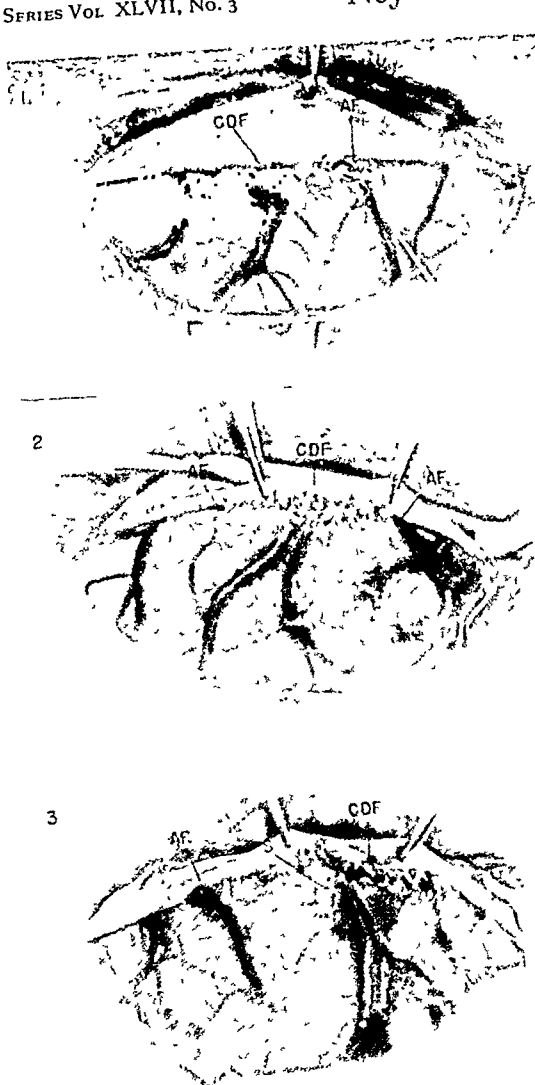
FIG. 3. Series of photographs taken at operation during exposure of the vertex in the surgical treatment of epilepsy. 1, AF, arachnoidal fistula through which bubbles of air are escaping after the local injection of air into the subarachnoid space. CDF, corticodural fixations corresponding to the motor area. Cerebrospinal fluid leaks into the subdural space through arachnoidal fistulas. Postural displacement of the brain occurs, and traction is thrown on corticodural fixations. 2, corticodural fixations in the nature of pacchionian granulations. Two arachnoidal fistulas, AF, are observed on each side of the fixations. 3, CDF, corticodural fixations corresponding to the motor area with partial obstruction of veins. S, scar of arachnoid. AF, typical arachnoidal fistulas.
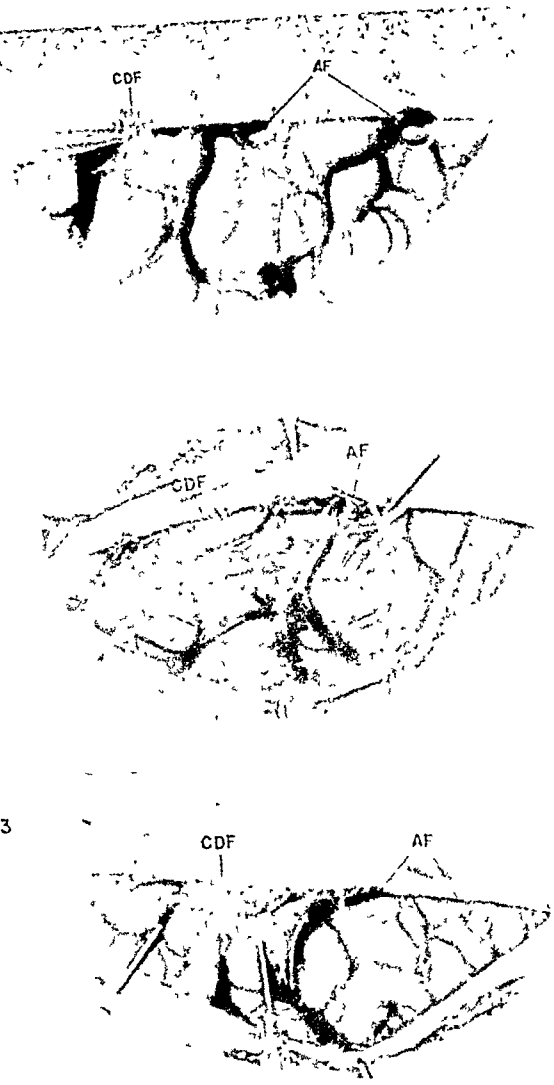
FIG. 4. Series of photographs taken at operation during exposure of the vertex in the surgical treatment of epilepsy. 1, AF, typical arachnoidal fistulas with thickened edges shown as the dura is gently lifted, exposing the cortex adjacent to the sagittal sinus. These fistulas are observed in all cases of epilepsy, and during operation there is a constant escape of cerebrospinal fluid through them. CDF, corticodural fixations with forceps passing under the scarred area which is located just posterior to the motor area. 2, typical corticodural fixations and a typical arachnoidal fistula. Arachnoidal fistula has thickened edges. 3, two arachnoidal fistula, AF. Corticodural fixations, CDF, binding the cortex, arachnoid, and dura in dense adhesions, are observed. The electrodes outline the motor area. Corticodural fixations are surrounded by an epileptogenous zone. Mild faradic stimulation of such an area produces clonic convulsions which pass to the other side of the body and continue after the electrodes are removed.

replaced by air will show the brain in postural stability; if, however, air has entered the subdural space and arachnoido-
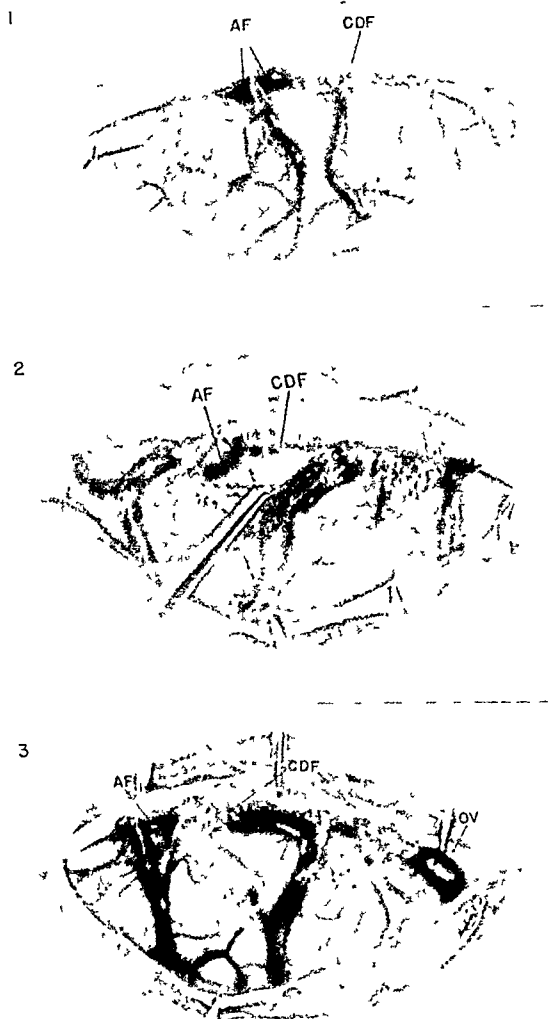

FIG. 5. Series of photographs taken at operation during exposure of the vertex in the surgical treatment of epilepsy. 1, two typical arachnoidal fistulas with thickened, edematous edges. Corticodural fixations are exactly over the motor area. 2, scar infiltrated arachnoid and cortex. Corticodural fixations partially separated, exposing the fistula. Arachnoid beginning to assume a typical jellyfish appearance. 3, a large arachnoidal fistula. Dense corticodural attachments. Obstructed vein, ov, due to corticodural fixations.

dural approximation is lost, cerebral postural displacement will be found to have taken place. X-rays show the displaced brain suspended by its attachments along the sagittal sinus, on which, apparently great traction stress is exerted.

The development of subdural air insufflation in the examination of the epileptic brain has made possible the roentgenologic demonstration of corticodural attachments at the vertex; subdural fluid collections; cerebral postural displacement; and actual visualization of the traction on corticodural attachments. It has served also to demonstrate certain factors which maintain cerebral postural stability and which hitherto have apparently remained unrecognized.

Subdural Air Insufflation. In 1932, I devised a technique for subdural air insufflation, to be used in the roentgenologic examination of the subdural space for the localization of areas of abnormal corticodural fixations. Subdural air insufflation has become a routine procedure in the examination of the epileptic in my service and has entirely replaced encephalography, although occasionally the two may be combined. The patient, with the back of the head shaved, is seated in a specially devised chair. Through a small burr opening in the occipital region the dura is nicked, and air rushes into the subdural space—it is not injected. For better visualization of the subdural space, from 3 to 5 ounces of cerebrospinal fluid are gradually withdrawn by an assistant while the skull is being perforated. Before the dura is nicked, the lumbar spinal fluid pressure in the sitting posture (normally from 24 to 30 mm. Hg.) is reduced as the spinal fluid is withdrawn. After 1 ounce is removed, the pressure is usually about 14 mm. Hg. After 2 ounces, the pressure is 6 or 4 mm. Hg. By this time the fluid is flowing through the needle very slowly. The dura is now nicked—air rushes into the subdural space, the lumbar pressure immediately rises to approximately 26 mm. Hg. (original pressure), and the cerebrospinal fluid spurts from the needle. This indicates that it is not the pressure of the cerebral fluid that maintains the brain in postural stability within the skull, since postural stability was maintained until air had entered the subdural space.

Fluid Film Adhesion between Arachnoid and Dura. Occasionally, during subdural air insufflation, after 1 or 2 ounces of

respirations, however, there is a sudden inrush of air, and the lumbar cerebrospinal fluid pressure rapidly ascends to about its
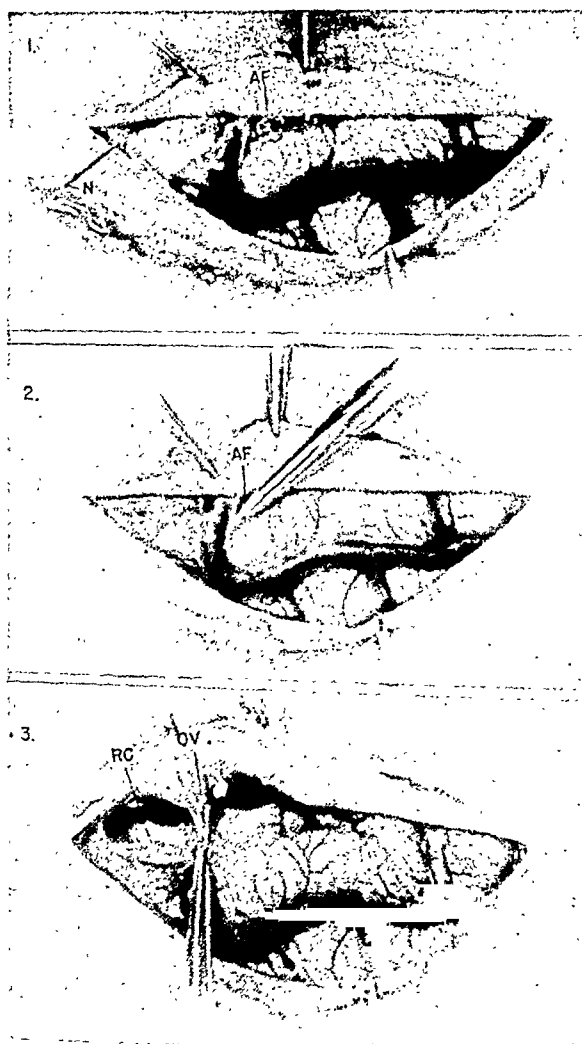

FIG. 6. Series of photographs taken at operation during exposure of the vertex in the surgical treatment of epilepsy. 1, N, local injection of air into the subarachnoid space with the escape of air bubbles through an arachnoidal fistula, AF. 2, forceps passed into the arachnoidal fistula, AF. 3, obstructed vein ligated. Scar obstructing vein has likewise obstructed veins passing along the saggital sinus. RC, retention cyst. Cavity sealed on all sides. Fluid collects in this cavity producing local cortical tension.
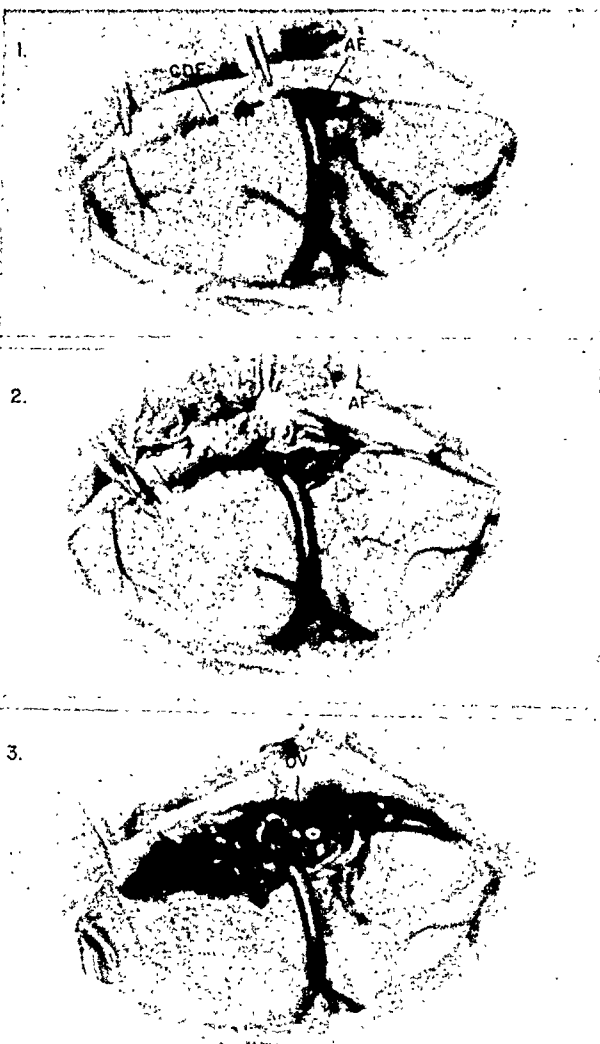
FIG. 7. Series of photographs taken at operation during exposure of the vertex in the surgical treatment of epilepsy. 1, dense corticodural attachments over the motor area with obstruction of veins. 2, area sealed with collection of fluid from a retention cyst. With separation of corticodural attachments, a definite cavity is found containing a number of varicose veins. 3, varicosity of veins paralleling the walls of the sagittal sinus is due to the obstruction of corticodural fixations. Just below ov will be observed the thickened edges of the arachnoidal fistula which loosely surrounds a cortical vein. This patient showed no characteristics of a local lesion. Was classified as, and had been treated for many years for, "idiopathic" epilepsy.

cerebrospinal fluid have been withdrawn by lumbar puncture and its pressure greatly reduced, a nick in the dura (it must be made completely through both layers of the dura) will not be immediately attended by the inrush of air which allows postural displacement of the brain, and the pressure remains very low. After a few deep

original level. When the dura was nicked, what maintained arachnoidodural contact when the cerebrospinal fluid pressure had been so greatly reduced? There normally

exists between the arachnoid and dura a thin film of fluid which has the adhesive properties common to fluid films. This fluid film adhesion between arachnoid and dura normally maintains them in apposition, and on this cerebral postural stability largely depends.

When fluid film adhesion between the arachnoid and dura is lost, as must occur when there is an excess of subdural fluid, there develops in the subdural space a negative pressure which maintains the brain in a certain degree of postural stability. This negative pressure, however, tends to syphon fluid from the subarachnoid into the subdural space through arachnoidal defects or fistulas, if such exist.

Subdural Negative Pressure. A demonstration of the negative pressure of the subdural space may be made during subdural air insufflation in the following manner: Lumbar puncture is done, removing 1 ounce of cerebrospinal fluid and reducing the pressure from, say, 26 to 14 mm. Hg. The dura is now nicked, air rushes into the subdural space as the fluid film adhesion between arachnoid and dura is disturbed, and postural displacement of the brain takes place. The lumber spinal fluid pressure ascends to its original height of approximately 26 mm. Hg. The burr opening in the skull is now firmly plugged with bone wax so that no air may enter. One or 2 ounces more of cerebrospinal fluid are withdrawn, again greatly reducing the lumbar cerebrospinal fluid pressure. The plug in the burr opening of the skull is removed, more air rushes into the subdural space, and the lumbar pressure again ascends with the further postural displacement of the brain. We see then that two factors are responsible for maintenance of the brain in postural stability within the skull: (1) the adhesive action of the normal thin film of subdural fluid which maintains arachnoidodural contact, and (2) a subdural negative pressure which, when film adhesion is disturbed, maintains the brain in a certain degree of postural stability.

Arachnoidal fistulas or defects assume particular importance when these facts related to cerebral postural stability are considered. The presence of an arachnoidal fistula predisposes to cerebral postural displacement, which takes place with an excess of subdural fluid. The maintenance of arachnoidodural film adhesion tends to seal arachnoidal fistulas, but the arachnoidal fistula is a weak point in the arachnoidodural adhesion and contact is easily disturbed. When an excess of fluid collects, cerebral support is then maintained solely by subdural negative pressure. This negative pressure syphons additional cerebrospinal fluid into the subdural space with an increasing degree of cerebral postural displacement. This condition, in which arachnoidal fistulas weaken arachnoidodural film adhesion, constitutes a condition of cerebral postural instability.

The exact outlet by which excessive quantities of subdural fluid escape is not known. We do know, however, that the straining of a convulsion or even that of vomiting, if the strain is sufficient to produce great cerebral congestion, tends to disperse excess subdural fluid, and arachnoidodural contact through film adhesion may then again be reestablished. This is apparently why convulsive seizures partake of their periodic nature, and why attacks so frequently occur in groups. It is not an uncommon occurrence for patients to go one or several months completely free of attacks and be apparently normal, and then suddenly, without apparent reason, have a number of attacks within one or several days. Very often a single convulsive reaction will suffice to remove this subdural fluid factor. Patients who have minor attacks, petit mal, seldom, if ever, have them in groups unless combined with major attacks.

Corticodural Attachments at the Cerebral Vertex. In a series comprising some 225 patients having the so-called "idiopathic" type of epilepsy, subdural air insufflation and surgical exposure of the cerebral vertex have been done. In practically all of the

patients in this series there were found at the extreme vertex of the brain, in or adjacent to the cortical motor areas, lateral expansions of corticodural fixations. These corticodural fixations very commonly occur where a large cortical vein approaches the sagittal sinus before turning to parallel the sinus. Occasionally these fixations are in the nature of pacchionian granulations with erosion of the dura and skull. In other cases these pacchionian features are absent but there is a dense corticodural fusion with thick and opaque arachnoid, and, when an attempt is made to separate the dura from the cortex, the pia is usually torn, leaving a denuded area. Very frequently extensive areas of corticodural fixations 2 or 3 cm. wide may be found binding the cortex to the dura and to the walls of the sagittal sinus, and often within these attached areas there are found large *varicosities* of the cerebral veins which have become obstructed by corticodural fixations as they parallel the walls of the sinus.

The corticodural attachments at the vertex in epilepsy are usually surrounded by a hyperirritable zone which *reacts* to stimulation with a convulsion. These areas will almost always react to weak faradic stimulation with a convulsion which spreads and continues after the electrodes are removed. If anesthesia is very deep, the convulsive reactions of the epileptogenous zones may be reduced, shortened, *or may* even be absent. These reactions are quite different from stimulation of the normal motor cortex in which there is a quick tonic reaction resulting usually in a *single* jerk which is maintained only during stimulation; normal reactions do not spread to other regions unless the stimulation be intense and prolonged, and cease immediately on removal of the electrodes.

Arachnoidal fistulas are very often found adjacent to these lateral expansions of corticodural fixations at the vertex. Occasionally the area around an arachnoidal fistula becomes sealed with adhesions and there forms a cavity into which fluid leaks and is retained. These retention cysts, found in separating corticodural attachments, usually have a soft gray base which is quite different from the scar of arachnoidal infiltration, both grossly and microscopically. Following trauma these corticodural attachments may be located in other regions, but wherever situated their visualization is made possible by subdural air insufflation.

The cortical veins approach the sagittal sinus more or less directly over the cerebral hemispheres, then turn to run forward some distance paralleling the sinus before fusing with its wall. This provision of nature tends to remove the stress on these veins during cerebral postural displacement, but in epilepsy, and possibly in other conditions, large cortical veins are caught in corticodural fixations and anchored so that they are subjected to the full traction stress of a cerebral postural displacement. So commonly do these corticodural fixations occur around large cortical veins at the vertex, that one is inclined to consider the cortical fixations an inflammatory reaction to traction stress, a reaction which ultimately may be so extensive as to obliterate the vein.

Increased Local Cortical Traction in Epilepsy. An extremely interesting observation was made by Leyton and Sherrington[2] in 1917: After ablation of portions of the brain in lower apes, collodion dressings were applied to the cortex and convulsions ensued; the removal of the dressings caused their cessation. These experimenters concluded that the convulsions were caused by traction exerted on the cortex with the drying of the collodion dressing. Foerster and Penfield[3] in a paper on traumatic epilepsy called attention to the induction of convulsions during operation by pulling on an adherent dura or by electrical stimulation of the cortex in the region of corticodural attachments. They state, "If the *increase* of a preëxisting strain produces an attack, it may well be that the preëxisting strain itself is an important factor in the etiology of spontaneous convulsions." Dur-

ing and after the World War I operated on many patients who had had compound craniocerebral injuries resulting in cranial defects which had developed into epilepsy; I found during operation that traction on corticodural attachments would almost invariably incite a convulsive reaction. The freeing of corticodural attachments would result in a cessation of attacks which recurred only if there were a reëstablishment of corticodural fixations.

In brain tumor we have a local increase of cortical tension due to the expansion of the tumor, and some types of tumor, in certain locations, are often accompanied by epileptic reactions. Frequently after such a tumor is removed, convulsive attacks will cease for six months or a year, but with the establishment of corticodural fixations (postsurgical), attacks recur even though there is no recurrence of the tumor—here in one individual we have two forms and two separate occurrences of localized traction with epileptic reactions.* If an increase of local cortical tension or traction is capable of inducing convulsive phenomena in cerebral neoplasms and in traumatic and inflammatory cortical adhesion, one may be justified in considering local cortical traction, if demonstrated in the "idiopathic" type of epilepsy, as an exciting factor. After subdural air insufflation, roentgenologic examination shows very clearly cerebral postural displacement with traction on corticodural attachments at the vertex. Examination, with the patient in the sitting posture and the head tilted at various angles, will reveal a great variation in the width of these attachments in different regions of the vertex. It is not uncommon to find lateral expansions of corticodural fixations extending 2 or 3 cm.

* The neurologic mechanism involved in convulsive phenomena I do not pretend to understand, but I feel convinced that it is not directly related to a local vasomotor reaction. I have frequently and carefully examined cortical vessels with a magnifying glass during the induction of a convulsion at operation, and have never observed any evidence of vasoconstriction, either by an increased pallor of the cortex, or in a diametric change in the smaller vessels.

from the midline in one location, while immediately anterior or posterior fixations may be very narrow; this local lateral area of corticodural fixations at the vertex is a focus of traction stress during abnormal movements of the brain, and around such a fixed area the cortex becomes hyperirritable—i.e., an epileptogenous zone is formed. The obvious question is, "Are these corticodural fixations abnormal?"—more particularly, "Is traction on them capable of inducing convulsive phenomena?" During subdural air insufflation (without anesthesia) when cerebral postural displacement was artificially induced, 74 per cent of these patients developed convulsive reactions or other type of epileptic manifestations within twenty-four or forty-eight hours, occasionally just after the dura was nicked. Traction, therefore, on these corticodural attachments is capable of instigating attacks.

Subdural air insufflation is accompanied often by headache, nausea, and vomiting, very much as in encephalography but to a lesser degree. The incidence of nausea and vomiting seems to retard convulsive reactions. To mitigate headache and convulsions, which tend to force out the air and subdural fluid, and to insure a more satisfactory roentgenologic examination, patients are usually given from 9 to 12 gr. of phenobarbital in 3 gr. doses immediately before the procedure. About 15 per cent of patients having subdural air insufflation under these relatively large doses of phenobarbital develop convulsive attacks.

CONCLUSIONS

The presentation of factors apparently active in the production of convulsive phenomena offers certain difficulties in the determination of causative relationship. Only the correction of these abnormalities with a cessation of epileptic phenomena would be the final proof of their causative nature. Two hundred seventy-two patients have been operated on in the endeavor to eliminate traction

on corticodural attachments at the vertex. Early evaluation of the results has been difficult because of a neurologic mechanism or conditioning process which develops in response to a long continued irritation. The irritation eventually results in an epileptogenous zone, and the epileptic reactions may require many months or years for their development, depending largely on the location of the lesion and intensity of the irritation. I have operated on a number of patients with well-localized cerebral traumas who developed attacks only after six, eight, ten years, or longer. There is good reason to believe that many children developing attacks during the second decade of life do so as a result of an injury sustained at birth. In such instances it is difficult to understand why the mechanism responsible for convulsive reactions was so late in developing, when a definite traumatic causative lesion had apparently existed for many years. It is not my purpose at this time to discuss this mechanism except to say that lesions in certain regions of the brain require a longer period to inaugurate the epileptic reaction. There seems to be a process of conditioning which neurologists look upon as a deteriorating of cerebral functional stability.

The question involved is, if it takes months or years for this epileptic reaction to mature or become conditioned to an irritant, will that mechanism immediately disappear on the removal of the irritation? If attacks should continue, though in diminishing form, after surgical intervention, does this mean that the lesion has been inadequately dealt with? A further complication is that most of these patients have become habituated to large doses of sedative drugs, particularly bromides and barbituates, and the sudden withdrawal of these after they have been used for months or years will usually induce in the epileptic profound psychic or convulsive reactions. We have had this occur repeatedly after operation, have resumed medication, and later have effected a complete but gradual withdrawal of medication with the cessation of attacks. In many instances, after the surgical correction of arachnoidal fistulas, the separation of corticodural attachments, freeing of obstructed cerebral veins, etc., there has been an immediate cessation of attacks; phenobarbital has been gradually eliminated; and patients have remained free from all epileptic manifestations. In some cases more than ten years have elapsed since operation. In others, recovery has been a process of gradual recession in the number and severity of attacks over several years. It would seem that in the latter group time and postoperative medication are necessary for the subsidence of the epileptic mechanism. It suggests that the epileptic zone surrounding a lesion, often requiring years to attain that degree of irritability sufficient to incite the epileptic reaction, will, in some cases, after apparently successful operation (determined by final results), require several years for its subsidence. The fact that certain patients are relieved of their attacks immediately after operation suggests that perhaps in the latter group the lesion was not entirely corrected. Before determining the value of any procedure in the surgical treatment of epilepsy, a number of years must necessarily elapse before results can be evaluated. In this series, in 87 per cent of patients from whom follow-up reports have been received, there has been a complete cessation of attacks or a reduction in the number of attacks by more than 90 per cent.

During the past ten years there have been several variations in the surgical technique used in dealing with traction at the vertex and cerebral postural instability. Analysis of cases grouped according to surgical technique indicates that better results were obtained by some of the methods employed than with others. Since it has been possible to locate arachnoidal fistulas and to deal with them directly, there has been a considerable increase in the number of immediate

or early cessations of attacks, particularly in patients whose onset of attacks had been fairly recent and who had not become habituated to sedatives. Even in the more advanced cases earlier recession of attacks is occurring. This experience suggests that perhaps the subsidence of irritability in the epileptogenous zone might have been more rapid in some of the earlier cases had the present technique then been in use.

This consideration of pathologic factors active in the production of convulsive phenomena in the "idiopathic" type of epilepsy is directed primarily to their consideration as causative agents; it is not my purpose at this time to consider the various surgical procedures used in their correction. That the surgical procedures used have been more or less successful in correcting these factors is obvious when one considers the results obtained. The correction of the factors herein considered has profoundly reduced all types of epileptic manifestations, and this, I believe, definitely indicates their causative nature. The pathologic condition may not always be the same, but, whatever its nature, the factor of local cortical traction seems to be one of great importance in the induction of convulsive phenomena.

While local cortical traction may be considered the exciting factor in the chronic convulsive state, the traction is made possible through those factors which are responsible for the cerebral postural instability, that is, the loss of fluid film adhesion between arachnoid and dura due to arachnoidal fistulas. The traction becomes intensified in local areas of the cortex which have undergone fixations to the dura, and it is quite possible that these corticodural fixations are due to the traction stress thrown on cortical veins at the vertex, which induces an inflammatory reaction with scar infiltration of arachnoid and pia. These fixations in some instances are undoubtedly due to trauma, possibly the tearing of veins passing through the subdural space resulting in collections of subdural fluid and blood. The principal

rôle of trauma, however, lies in arachnoidal tears which, failing to heal, become permanent fistulas through which fluid periodically leaks into the subdural space.

SUMMARY

1. In a series of 272 patients with the so-called "idiopathic" type of epilepsy, certain definite lesions have almost invariably been found at operation, the more common types being shown photographically.

2. In some 225 of these cases, corticodural fixations at the cerebral vertex in or near the cortical motor areas were localized roentgenologically by subdural air insufflation, in which cerebral postural displacement was artificially induced by allowing air to enter the subdural space.

3. Arachnoidal defects or fistulas through which cerebrospinal fluid leaks into the subdural space have been found in practically every epileptic patient operated on since their characteristics have been recognized. These fistulas are probably the result of tears in the arachnoid which have failed to heal. They are usually located at the extreme vertex of the brain where the strain of gravity is greatest.

4. During subdural air insufflation it is possible to demonstrate that cerebral postural stability is dependent on the maintenance of arachnoidodural approximation. This approximation is controlled by two factors: (1) fluid film adhesion between arachnoid and dura which maintains these structures in forceful apposition; (2) a negative subdural pressure which develops when fluid film adhesion is lost through an excess of subdural fluid. This subdural negative pressure maintains the brain in a certain degree of postural stability but tends to syphon cerebrospinal fluid into the subdural space through arachnoidal fistulas when such are present.

5. The traction on the cortex which occurs in expansive lesions and corticodural fixations after craniocerebral trauma seems

to be a factor responsible for the induction of convulsive phenomena in these two diverse pathologic conditions. During operation traction will serve to induce attacks. In the "idiopathic" type of epilepsy definite traction is exerted on corticodural attachments at the extreme vertex of the brain during postural displacement which occurs when there is an excess of subdural fluid. Subdural air insufflation, devised for the examination of the subdural space, serves in the localization of corticodural attachments; it shows the cerebral postural displacement which takes place when air or excessive fluid enters the subdural space; subdural fluid is determined by a definite fluid level which changes with position of the head.

6. In this series the surgical correction of arachnoidal fistulas and the reduction of traction on corticodural attachments have so diminished convulsive attacks in the epileptic that one is justified in considering the lesions which are herein presented as definite causative factors in the chronic convulsive state.

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TREATMENT [of septic wounds] has radically altered since the days of the war of 1914–18. The advocates of irrigation now recognize that "it is the mechanical washing far more than the action of the antiseptic that is responsible for any improvement." . . . I doubt if irrigation will long survive at all.

From—"War Wounds and Air Raid Casualties" (Lewis).

THE RELATION OF CERTAIN SEMINAL FINDINGS TO FERTILITY, WITH SPECIAL REFERENCE TO SPERM CONCENTRATION AND THE SIGNIFICANCE OF TESTICULAR EPITHELIAL CELLS IN SEMEN

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I T is axiomatic in nature that the soil determines the crop, both as regards quantity and quality. In the same way testicular function is evidenced by the seminal picture. In 1936 I reviewed before the New York Obstetrical Society the results of the morphologic and biometric studies of the semen during the previous ten years.[30] I naturally could not cover the entire subject because of lack of time. Thus I could not go into the subject of sperm concentration, nor consider the significance of certain epithelial cell forms found in the seminal fluid. These cells I have studied especially during the last three years.

I have pointed out repeatedly that most of the reports on the relation of oligospermia to fertility were of no value because they failed to take into consideration the morphologic and biometric characteristics of the spermatozoa and thus did not separate oligospermia with normal sperm from oligospermia in which the poor quality of the sperms would in itself account for the infertility present.

I. THE RELATION OF THE NUMBER OF SPERMS TO FERTILITY

The opinions regarding the number of sperms necessary for fertility vary tremendously. Thus the normal number of sperms per c.c. for man is believed by many to lie around 100 million whereas other authors accept a much lower figure. I have examined and counted many semen specimens and 100 millions per c.c. seems to me to be too high as an average. With a Leitz microscope, a number 6 objective and a 10X ocular and the usual tube length (140 mm.) the approximate area of the visible field is $\frac{1}{10}$ square mm. If a thick drop of semen is used and a thin cover glass the approximate thickness of the seminal layer will be 0.1 mm. A short calculation will show that one sperm to a field will equal 100 sperms per c. mm. or 100,000 per c.c. Where 100 million sperms per c.c. are present, each microscopic field would show 1000 sperms, certainly a picture beyond the average.

The above described method of determining the number of sperms per c.c. is accurate enough for all general purposes. Even the results of instituted treatments can be so evaluated since normally semen specimens from the same individual under apparently identical conditions vary quite considerably. For accurate work, of course, a counting chamber must be used (hemocytometer). As many fields as possible should be counted (I count all the 400 small squares) since an even distribution of the sperms is not easy to attain.

It is well in all cases (except where very few sperms are present) to dilute the semen, since its density may otherwise make a bothersome focussing up and down necessary to see the sperms at the various levels of the 0.1 mm. deep counting chamber. I personally prefer a 1 per cent chlorozene solution as a diluent since it kills the sperms and also dissolves most of the mucus present. Specimens of semen with good sperm concentration have to be diluted and the dilution must vary according to the sperm concentration present in the particular specimen. A dilution in the gross is much preferable and more accurate than pipette dilution. If desired a coloring

agent such as methylene blue or cresyl violet may be added.

In thus counting the number of spermatozoa per c.c. what may we interpret as oligospermia of sufficient degree to influence fertility? Certainly, as many authors have pointed out, as I have for man[10] and Polozow[33] and Polozow and Nagajew[34] for the stallion, the capacity for the production of spermatozoa varies very much in different individuals without there being any evidence of disturbed fertility. I feel sure that Macomber and Saunders[35] have disregarded other factors which might have caused infertility when they claimed that fertility was practically never present with sperm concentrations of less than 60 million per c.c.

The same authors believe that it is the sperm concentration and not the total number of sperms present in the ejaculate which is the important factor. While the importance of sperm concentration cannot be denied (Cohn,[36] Fuchs[37]) the total number of the spermatozoa seems to be of far greater importance. Hutt,[38] for example, has shown for the rooster that high sperm concentrations do not mean high fertility. The least fertile of his roosters had twice as many sperms per c.c. as the one with the highest fertility, and Hutt states: "Fertility in the male fowl is dependent upon physiological efficiency of the spermatozoa rather than upon their quantitative production."

Lloyd Jones and Hays[39] found that in a male rabbit twenty services in four hours decreased the number of sperms from 35.6 millions per c.c. to 300,000 per c.c. and the fertility from 72.09 per cent to 35.55 per cent. But the fact that even with the latter figure conception still occurred in about one-third of the cases shows that here again nature is very lavish and allows very great reduction from the optimum before she admits defeat.

Walton,[40] taking sperm from the epididymis of rabbits, found that fertility was definitely reduced by dilution of the semen with normal saline to a concentration of one

million or less and that sterility was present when the sperms averaged less than 10,000 per c.c. It is to be noted, furthermore, that in these experiments the spermatozoa were taken from the epididymis and so probably were not fully matured. In addition, the diluent changed environment, buffer reaction, osmotic tension, etc.

Iwanow, Kusnetzowa, Milowanow, Neumann, Nagajew, Skatkin[41] used bull sperm, diluted thirty-two times, for artificial insemination with very good results. These authors state that they feel that even a much greater dilution could have been used satisfactorily.

Lagerloef[42] in his exhaustive monograph on the sperm morphology of the bull, which he compared directly with the pathologic findings in the testes in many cases, says: "My investigations show that the usual cause of decreased sperm production is a degeneration of the seminal tubule epithelium. Whatever the agent is which produces these changes it causes in most cases a disturbance of spermatogenesis as a whole and not only a decrease of the number of sperms."

We see then that a very great reduction of the number of sperms can occur before actual sterility supervenes. My own cases fully corroborate this. I will cite here only two of them.

CASE I. M. M., a white male, 40 years old, married five years. His wife, 30 years old, was a hypothyroid. No contraceptives had ever been used. Three spontaneous abortions had occurred at three to four months of gestation, the last one two and one-half months before examination.

Semen specimen was about 2.5 c.c. in amount, slightly increased in viscosity. Motility was good and continuous for many hours in about half of the sperms. The total number of the sperms was 1,800,000 per c.c. Sperm morphology was slightly above the normal limit: 24 per cent head changes, made up of 9 per cent small heads, 1 per cent large, 1 per cent tapering, 9 per cent round and 4 per cent miscellaneous. Since round heads are in most cases not of great significance I feel that probably the hypothyroidism of the wife

played a role in the abortions. Other endocrine imbalances may also be important but the wife so far has not been willing to have the proper examinations carried out.

I have emphasized before[14, 30] the difficulties of determining motility of the spermatozoa accurately; it is not necessary to go into this question here. I do, however, want to state that I believe the determination of the percentage of motile sperm in any specimen to be a very haphazard procedure. Often one sees a sperm, characterized by some peculiarity actively motile, and the same cell absolutely immotile a few minutes later and later again we find the same cell with good motility. What is true of one sperm is of course true of others also. Some semen specimens from known fertile men, because of the effect of some almost imponderable agent or agents, will show only a relatively small number of motile spermatozoa.

The second case may illustrate this point as well as definite oligospermia with normal clinical fertility.

CASE II. G. M. Semen (friction specimen in office) 2.5 c.c. in amount was grossly normal, with one million sperms to the c.c. Very active motility of apparently less than half of the sperms was present. Morphology and biometrics were normal. The clinical fertility of this man was very high.

I agree with Macomber and Saunders[35] that small testes usually produce fewer sperms than large normal ones, but I cannot agree with them in believing that nature will function only in such narrow limits as are represented by the difference between 60 and 100 or more millions. I subscribe to the opinion of Hutt[36] that fertility depends not on the quantitative production of the sperms but on the physiologic efficiency of those produced.

I still feel as I did years ago that if twenty-five to fifty otherwise normal sperms are present in each high dry power microscopic field, this number should be sufficient to insure offspring, at least under the conditions existing among humans. I want to emphasize, however, that motility does not indicate the fertilizing power of the spermatozoon.

Perhaps some readers have also expected here a consideration of the hydrogen ion concentration of the semen. However, as this is usually carried out, it is of no value, and investigations by many workers have shown that the buffer reaction of the seminal fluid is such that in most cases the sperms are well protected for many hours and minor variations of the hydrogen ion concentration mean little or nothing.

II. SOME NOTES ON SPERM MORPHOLOGY ESPECIALLY ON THE SIGNIFICANCE OF CERTAIN CELL FORMS FOUND IN THE SEMEN

In previous reports I have discussed and pictured various sperm abnormalities to the number of about fifty. Some authors since then, especially Belding,[43] Generales and Stiasny[44] have gone much further and have divided the various morphologic sperm forms into hundreds of types. This to me seems an unnecessary complication of details. It is self evident that one can divide the large sperm heads into large round, narrow, tapering, irregular, and add differences in staining reactions. Any one of the other main types of sperm head forms can also be thus split up into subtypes. The result will be the tabulation of an enormous number of different forms which will, however, not lead to any clarification of the subject. On the contrary, it will only lead to confusion.

In the morphological evaluation of the spermatozoa I have always advised that one take the most prominent feature of the sperm and sperm head as the criterion, as for example, large, small, narrow, etc. If we divide all our groups into subgroups the result will be not only that the fine differences will make it impossible to agree with ourselves in recounting the same specimen but the individual groups will be so small that their proper meaning will be next to impossible to determine. Aside from this,

there is great danger that, looking for too fine distinctions will cause variations which should still be considered normal to be tabulated as abnormal. The difficulty in evaluating any semen specimen as to its fertilizing powers lies not in picking out definitely narrow, large, small cells, etc., but in knowing where to draw the line between normal and abnormal.*

For the average report of the morphologic characteristics of a semen sample I use only the classifications: large, small, narrow, tapering, round and miscellaneous head forms. The last group includes thickened end knobs, aberrations of the staining reactions, amorphous forms, etc. The body changes are classified as thickened body, coiled, double, and miscellaneous—which last group includes such forms as abaxial implantation, naked body fibril, etc. Usually the cell forms grouped under miscellaneous are so few in number that reporting them on a percentage basis would give a false impression. If, for instance, one sperm in 500 is found with an abaxial implantation one cannot say that $\frac{1}{5}$ per cent of the sperms are abaxial because the next thousand cells or more may not show a similar change. Should, however, one of the sperm forms listed under miscellaneous be quite frequent in a semen specimen, then such forms are taken out of the miscellaneous group, placed under a specific heading, and specially noted, just as the presence of certain immature forms and cells of spermatogenesis are separately indexed. Of these forms I will speak later.

I do feel, however, that while in the classification cited, all narrow and all tapering sperm heads should be grouped together one should in addition distinguish between long and short narrow and tapering forms. (Fig. 1.) I have always stressed the sinister import of these narrow and tapering sperm heads and have made special notes on their presence, especially of the very long heads and the sickle-shaped ones. (Figs. 2, 3 and 4.) The proof that these head changes are really very important has been given by Lagerloef[42] in the bull. This author examined both semen and testes of bulls and found that tapering and what he calls pear-shaped heads are especially frequent in severe types of testicular degeneration.

Aside from the sperms themselves there are, however, other cell elements present in the semen. To demonstrate them the cleared smears used for sperm morphology are of no value. I use thin smears quickly dried in a current of air and stained without fixing if the stain used contains alcohol. If it does not then 50 per cent and 95 per cent alcohol is used successively as a fixative. Good results can be obtained with an old (polychrome) methylene blue, or with eosin and methylene blue. The carbol-fuchsin-eosin stain I usually employ for the spermatozoa may also be used.

The non-spermal cells of the semen can be grouped under three headings:

1. Red and white blood cells. Such cells may very occasionally be found in normal semen, but when present in greater numbers red blood cells and leucocytes, especially in clumps, denote congestion and inflammation. The approximate site of such an inflammation may at times be diagnosed by the type of the other cell elements present, such as urethral, prostatic or testicular cells. Since the meaning of red blood cells and white blood cells as well as

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* My own investigations and those of others so far seem to show that a proper evaluation of the sperm morphology and biometrics in all their aspects allows conclusions to be drawn about the fertility of the respective individual. My own attempts to disprove this hypothesis have always been unsuccessful. In J. A. M. A., 112: 1817, 1939, Frances I. Seymour published a report of a case in which only 3 per cent of the sperm heads were abnormal in a semen specimen containing 94,000,000 sperms per c.c. of good motility. No mention is made of the biometric findings and perhaps these were abnormal. Morphology and biometrics usually run parallel, but this is not necessarily so, as I have pointed out before. I have never seen any case with only 3 per cent abnormal head forms among hundreds of normal seminal specimens with good clinical fertility. Even a normal bull, where the head changes are less frequent than in man, generally has more than 3 per cent abnormal head forms. I seriously think that the morphological count must have been made a little too liberally. Arrangements have been made to check this case and it will be reported upon later because of the importance of the question to be settled.
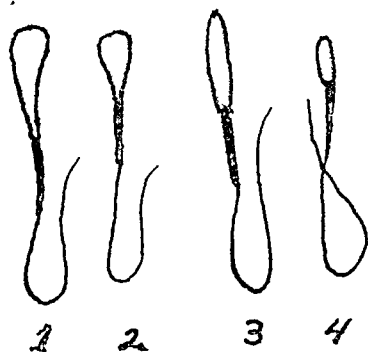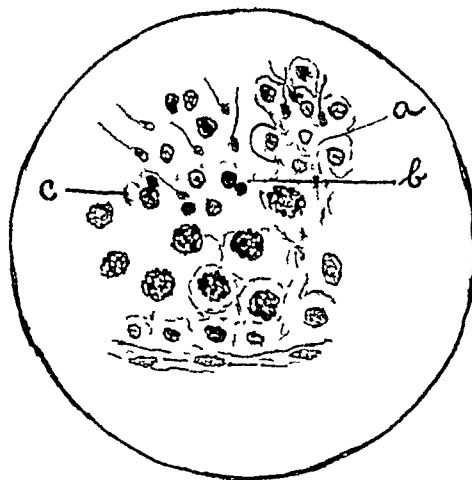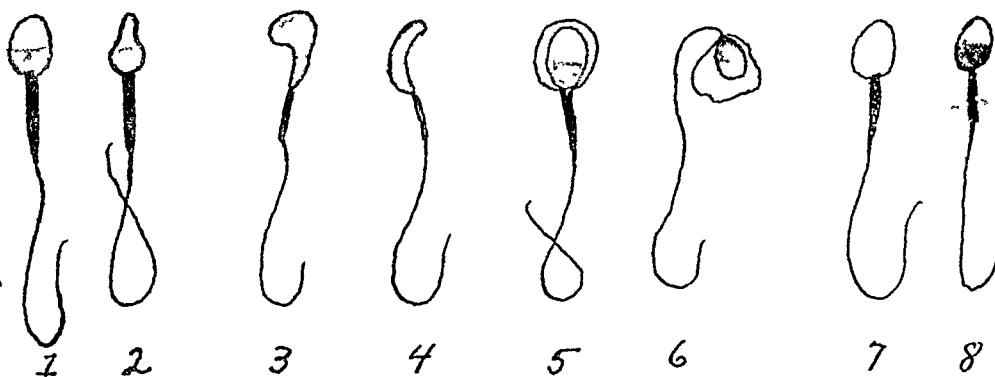
FIG. 2.

FIG. 1. Cells 1 and 2 represent tapering sperm heads and cells 3 and 4 narrow sperm heads. Sperms 2 and 4 are the usual forms and 1 and 3 very long types which are especially indicative of severe spermatogenic disturbance. Often the first sign of improvement in a semen specimen is a shortening of these very long forms, resulting of course in a decreased value of the coefficient of variability while the morphological cell count still remains the same. (See text.)

FIG. 2. Sperms 1 and 2 are normal forms (cell 2 seen from the side). Sperms 3 and 4 are sickle-shaped heads, like the tapering and narrow forms, of definitely sinister import. Sperm 5 still has a thick mass of granular cytoplasm (spermatid rest) around the head. This cytoplasm stained much darker than normal. Sperm 6 also has an irregular cytoplasmic mass around the head. There is a tail but no body present. Sperm 7 is an almost unstained cell head. I believe these sperms are old and degenerating forms which are being cast off. They seem to be more frequent after prolonged periods of sexual rest. Sperm 8 is normal except for some mucus adhering to the body. I believe this to be of no significance.

FIG. 3. Portion of seminiferous tubule of a human, adult testicle. From below upwards we see the tubule wall with light staining, elongated nuclei. Then a single layer of spermatogonia. Above these are two layers of spermatocytes with large nuclei with the chromatin in a skein-like formation. Above the spermatocytes are the spermatids, smaller cells which form the spermatozoa. a, a sertolian cell. This with the attached spermatids and the embedded spermatozoa forms the so called spermatoblast. b and c, spermatids with two nuclei. The one at c also shows a vacuole. (Drawn semischematically at a magnification of 400 diameters.)

their cytology is well known it is not necessary to discuss them further.

2. Epithelial cells from the male tubular system, tubuli recti, vas deferens, prostate, urethra or urinary tract itself. Here we find columnar, transitional and squamous epithelial cells. Their cytology also is well known and needs no discussion here. Any one of these cell forms may be found singly in normal semen but when present in greater numbers or in connected sheets they naturally denote irritation of some sort. It might be well here to mention also the corpora amylacea of the prostate, laminated in appearance and staining with iodine. These bodies are found especially in exhaustion and with increasing old age when they may form concretions up to 1 mm. or so in diameter. Usually they are 10 to 30 microns in size.

3. Cells from the testes themselves (seminal tubule epithelium). Some of these cells, for instance spermatids, are occasionally found in normal semen ejaculates but their numbers and types are much increased in abnormal cases. All these cell forms have been described years ago by Broman,[45] Stieve,[46] and others, but it is their diagnostic significance in semen evaluation that I wish to stress here. In order to understand this significance it is necessary to review briefly the spermatogenetic processes.

The seminiferous tubules, invested with a thin connective tissue membrane, show a very characteristic epithelial lining which can be divided into three different groups of cells. (Fig. 3.) On the basement membrane lies a single layer of rather small cuboidal spermatogonia. These divide to form spermatogonia and spermatocytes of the first order, rather large cells with very large nuclei and with a skein-like arrangement of the chromatin. From these the spermatocytes of the second order are formed and these in turn form the spermatids, small spherical cells with little cytoplasm and a large spherical nucleus showing irregular karyosomes. In this process the spermatocytes of the second

order each form two spermatids, presumably by amitotic division, so that the number of chromosomes is reduced to one-
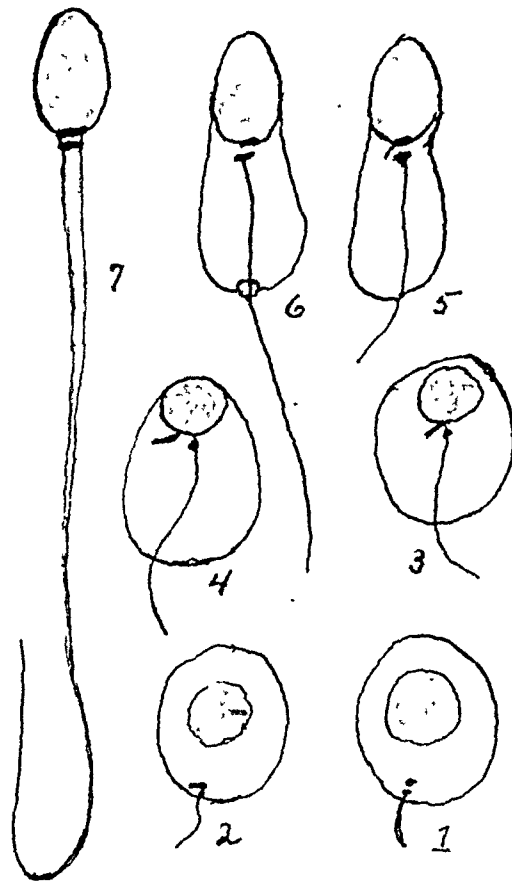

Fig. 4. Human spermatogenesis (schematic according to Meves, Bonnet). The division of the centrosome, formation of the end knobs (later in the base of the sperm head), tail and end ring are shown. Also the gradual protrusion of the sperm head from the cytoplasm of the original spermatid. (Compare with Fig. 6.)

half of the original number. The spermatids then change to spermatozoa. (Fig. 4.)

It is only in the spermatid that the centrosome divides and a tail grows from the posterior half. Such cells as Carey and Hotchkiss[47] present as spermatocytes are therefore not spermatocytes but abnormally developing spermatids of the type I have pictured before. (I might add here also, not in a spirit of criticism but simply to avoid someone's being led astray, that I cannot agree with them on their Figure 1 which they label normal spermatozoa. If the top cell is normal then the lowest one is certainly too small, or vice versa. The

central sperm is long and narrow, with a definite taper and seems to have an abaxial implantation.)

double nuclei and with vacuoles are seen. (Fig. 3.)

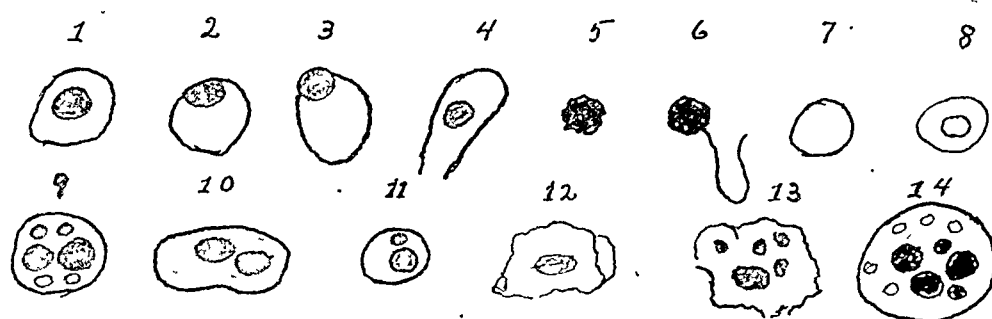Spermatid giant cells, evidently caused

FIG. 5. Cell 1 is a spermatid with a rather excessive amount of cytoplasm. Cells 2 and 3 are spermatids with the nucleus at the periphery and protruding. Number 5 is an extruded spermatid nucleus, number 6 one with rudimentary tail. (Compare with number 1 in Fig. 6.) Cell 4 may be part of the sertolian cell system, though probably it is a deformed spermatid. Number 7 is a protoplasmic drop. Such drops are frequently seen in sizes from 2 to 10 and more microns. Cell 8 is a degenerating spermatid, the nucleus of which remains practically unstained. Cell 9 is a giant multinuclear spermatid with vacuolization. Cell 10 is similar to 9 but has no vacuoles. Cell 11 is a normal sized binuclear spermatid. Cell 12 a squamous epithelium cell from the urethra or skin. Cell 13 apparently has been torn loose from a spermatoblast, for it contains several sperm heads. These cells some authors have interpreted as macrophagocytes but Stieve[46] believes that they are the result of cell fusion "sperm agglutination." I believe in some cases similar pictures are produced by active penetration of the spermatozoa into the cell. (See text.) Cell 14 is a huge, multinuclear, degenerating spermatid with vacuoles.

Besides the spermatogonia, the spermatocytes and the spermatids in the testicular tubules there are also present the so-called "sertolian" cells, large and irregular in form, which arise from the primordial cells. They grow upward like bushes, unite with the spermatids and are the embedding medium for the developing spermatozoa which break away when their development is completed. The union of the sertolian cell and the spermatid forms the "spermatoblast."

Since testicular epithelium is very sensitive, aberrations from the described and pictured development often occur. In fact Kyrle,[48] Schinz and Slotopolsky,[49] Romeis[50] and others have always found in boys and adult men evidence of atrophy in some of the seminiferous tubules. They consider such localized atrophy as part of the normal picture. Although Stieve[46] denies this, I myself have generally seen some tubules in an apparently normal human testicle which showed no spermatogenesis and at times some degeneration (perhaps temporary). Occasionally spermatids with

by fusion, are at times found and although these cells of course indicate degeneration, the rest of the slide may seem normal. When such cells lie loose in the lumen of the testicular tubule, as they frequently do, they of course may be expelled with the semen. However, Lagerloef[42] states that while he has seen such cell forms in the epididymis and even in ejaculated bull semen, he has never seen spermatocytes or spermatogonia there. This in general has been my experience also, although spermatids are not infrequently found in both normal and abnormal semen specimens. It is true, however, that most of these show some more or less marked evidence of degeneration.

Cell 1 of Figure 5, for example, seems too large; and cell forms such as 2 and 3 are certainly not normal. At this stage, when the nucleus is at the periphery, a comparison with Figure 4 shows that evidence of tail formation should be present. Sometimes such forms as 5 and 6 are also seen in the semen. Number 5 is evidently an extruded spermatid nucleus and number 6

even has a rudimentary tail but no body (compare with number 1 in Figure 6). Many semen specimens also show vesicular

With one exception, to be described later, I have, like Lagerloef[42] never seen spermatocytes or spermatogonia in human
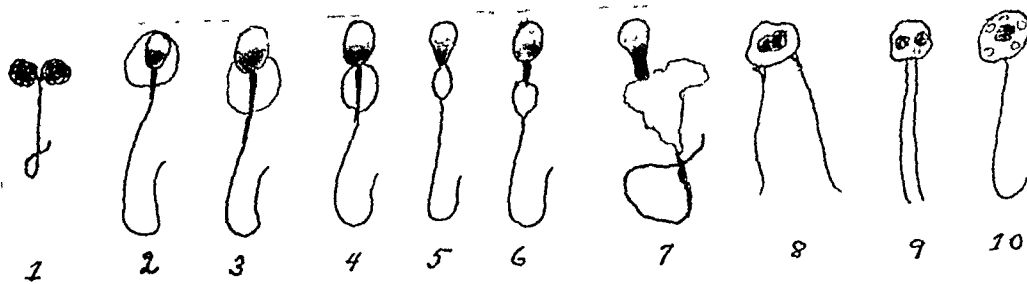

Fig. 6. Number 1 is an extrusion of the nuclei of a binuclear spermatid. There is a rudimentary tail but no body present. Sperms 2, 3, 4, 5 and 6 show various unripe forms in which the gradual casting off of the cytoplasm can be seen. (Compare with Fig. 4.) Cell 7 apparently was derived from a giant spermatid of which the large amount of cytoplasm still clings to the cell. Cells 8, 9 and 10 are abortive attempts at spermatozoal formation. Cell 10 shows vacuoles.

cytoplasmic masses of varying size (number 7 in Figure 5). They represent either a spermatid which has extruded its nucleus or cytoplasmic drops cast off by the developing spermatozoon. (Figure 6 shows these various stages of the casting off of the cytoplasm.) Such cytoplasmic masses are found not infrequently in small numbers in normal semen. They increase markedly with sexual overloading. In degenerative testicular processes they are very numerous in the early stages, but decrease later on. They again appear when regeneration sets in. Numbers 8, 9 and 10 are very abnormal and degenerating spermatids; 9, 10, 11 and 14 show multiple nuclei. Some also show degeneration vacuoles. Cell number 4 of the same figure approaches the shape of a sertolian cell, but I believe it to be a spermatid broken off a spermatoblast. The same is true of number 13 which shows various embedded sperms. Of course one must think of fused spermatids here also, that is, sperm agglutination in the sense of Stieve.[46] Another possible explanation is that active penetration of the spermatozoa (such as I have described in a lymphocyte[30]) may have taken place.*

* After the present article was written I received two reprints from Dr. Ch. Joel (Michael and Joel: Zellformen in normalen und pathol. Ejakulaten und ihre klin. Bedeutung. Schweiz. med. Wcbnschr., 67: 757, 1937 and Pollak and Joel: Zur Morphol. d. maennl. Keimzellen. Arch. f. exper. Zellforsch., 22: 77, 1938). These authors believe that these cell forms are evidence

semen. Furthermore, I have never seen any single testicular epithelial cell which I could definitively identify as a spermatocyte or spermatogonial cell.†

It seems to me that such cells could be loosened from their bed only in the severest types of testicular degeneration. In such an event, the cells themselves would be so degenerated that they would probably be absorbed before they completed their passage through the long tubular system of the male genital tract, or if really ejaculated would have become unidentifiable. Furthermore in testicular degeneration the spermatocytes are the first to show degeneration—cystic in type—as shown so beautifully by the work of Lagerloef.[42]

Interesting as the study of these testicular cells is, I want to stress here particularly their clinical significance when present in ejaculated semen. I have pointed out before that the difference between finding no

of spermatophagia which they think they have actually seen. I cannot argue this point, but I have never seen anything to suggest such spermatophagia and must consider the explanation given in the text as more probable.

† The authors cited in the previous footnote claim to have found spermatocytes and even spermatogonia in many abnormal semen ejaculates. This may be so, but their pictures of such cell forms do not seem convincing. It has been pointed out by many authors (Schinz and Slotopolsky,[49] Stieve,[46] and others) that the various cell forms of the testicular tubular epithelium are often very hard to differentiate. This is especially true when such cells occur in the ejaculated semen where they are nearly always degenerated.

sperms at all and only one or two is not just a few sperm cells. In the first place the finding even of one single sperm shows that at least some sperms are produced and in addition precludes occlusion of the excretory seminal ducts. Thus a puncture of the testicle to determine the presence or absence of spermatozoa in this organ becomes unnecessary. In the same way the presence of any of the testicular cell forms—forerunners of the spermatozoa—in the semen excludes occlusion of the seminal passages.

Testicular cells in the semen have a greater significance than that just mentioned. Given all the data of the case and the seminal picture, it is frequently possible to distinguish between certain forms of disturbed spermatogenesis and to evaluate the effect of a given treatment. A normal semen will show a very occasional spermatid and some protoplasmic drops, but I have not found more than one to five spermatids per 1000 spermatozoa counted in a normal specimen. The protoplasmic drops, while they are more frequently encountered than the spermatids, should not be abundant, since they occur mainly when unripe spermatozoa are ejaculated. Thus sexual overloading will lead to an increase of these protoplasmic drops. Generally the picture is completed by the presence of many unripened, coiled and improperly stained sperm heads. Such a picture might also be present in testicular hypoplasia, but the clinical data and rechecking of the seminal picture later on often make a differential diagnosis possible. Protoplasmic drops would naturally also be found in the early stages of acute testicular degeneration as Lagerloef[42] has shown in the bull. In addition one would also expect to find in such cases many long narrow and tapering and sickle shaped sperm heads and often almost a complete résumé of sperm pathology. A case from my practice may illustrate this:

CASE III.   H. N., male, white, 36 years old, came to me because, though he had been married eleven years, there were no offspring. Physical findings were negative, the testes being of average size and firm. The semen showed only about 50,000 sperms per c.c., and motility was only fair. The seminal morphology showed 50 per cent abnormal sperm heads with many narrow and tapering forms and an increased number of spermatids and protoplasmic drops, indicating as I believe, hypoplasia of the testicle and degeneration, at least in some areas.

Under treatment the patient improved. Three months later the amount of the semen ejaculate had definitely increased and the number of the sperms had risen to more than 1,000,000 per c.c. while the motility was much better. The abnormal sperm heads now totaled only a little over 30 per cent and there were fewer of the long tapering and narrow forms. The sickle-shaped heads had disappeared. Spermatids and protoplasmic drops had increased in number (evidence of regeneration).

Shortly after the last semen examination the patient contracted mumps with bilateral orchitis. Three weeks later the semen showed only about 10,000 sperms to the c.c. The motility was very poor. Most of the sperm heads were now abnormal with many very long narrow and tapering forms. Many protoplasmic drops and degenerated spermatids, often multinuclear, were found. The picture was that of an acute testicular degeneration.

As noted in parenthesis above, protoplasmic drops may also mean regeneration, either spontaneous or the result of treatment. In such cases where new sperms are being formed, many of them will at first be unripe and thus the number of the protoplasmic drops may indicate the effectiveness of the treatment, showing that active regeneration is taking place.

CASE IV.   F. P., white male, 30 years old, showed in the first semen specimen only 25,000 sperms to the c.c. with a few spermatids and protoplasmic drops. Under treatment the sperms increased to almost 3,000,000 to the c.c., while the number of protoplasmic drops about equalled the number of spermatozoa.

Spermatid giant cells and parts of the sertolian cell system always indicate degeneration in at least some of the tubules

of the testicle and the extent of such degeneration can be judged by the proportionate number of such cell forms seen. The presence of spermatocytes and spermatogonia would be a sign of further rapid and ultimate degeneration in the tubules of the gonad. Such a condition was present in Case III at the second examination made two months after the attack of orchitis. The semen was then only about one-half of the previous amount. Only one deformed immotile spermatozoon was found after a long search. Protoplasmic drops were practically absent, but degenerated spermatids, spermatid giant cells, many multinuclear and vacuolated, were seen. This is the only case in which I have seen spermatocytes and spermatogonia that I could identify definitely. The testicular tubule epithelium in some instances was cast off in sheets and the relative position of the various layers of the seminiferous tubule epithelium, though degenerated, could still be recognized.

Besides the seminal pictures described, there are also other semens which show few sperms with poor sperm morphology and practically no spermatids or protoplasmic drops. In such cases we are generally dealing with chronic testicular degeneration such as occurs in wasting diseases or is present in older men where spermatogenesis is definitely reduced.

In conclusion, I wish to emphasize that the evaluation of the sperm pictures as presented is, of course, only tentative and needs much more investigation before really definite answers can be given. Should it become possible to correlate various seminal findings and clinical lesions a most important advance would have been made from the diagnostic as well as the prognostic standpoint.

SUMMARY

The number of sperms produced ordinarily is far in excess of that needed to fertilize the ovum.

Very long, tapering and narrow sperm heads are of especially sinister import and

their shortening is often the first sign of improvement.

By evaluating the clinical data, the sperm morphology and the presence and number of the protoplasmic drops and seminiferous epithelial cells, it is frequently possible to distinguish between sexual fatigue, testicular hypoplasia and acute and chronic testicular tubule degeneration.

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COMBINED INTRA-UTERINE, EXTRA-UTERINE PREGNANCY*

WITH A REVIEW OF 291 CASES IN THE LITERATURE AND A REPORT OF THREE NEW CASES

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COMBINED pregnancy, i.e., simultaneous extra-uterine and intra-uterine pregnancy, is not so rare as many writers would lead one to believe. Parry,[25] in his monograph in 1876, stated that "twenty-two of the 500 cases of tubal pregnancy collected by him, were complicated by a coëxisting pregnancy."

The simultaneous occurrence of gestation within and without the uterus is, of course, a type of twin pregnancy, one fertilized ovum implanting itself in the uterus and one in the tube, or perhaps rarely in the ovary. The cause of this condition is some interference with the passage of the fertilized ovum from the ampulla of the tube, where fertilization usually occurs, into the uterus. Conception need not necessarily take place in the tube, for the spermatic particle may advance as far as the ovarian fimbriae to meet the ovum or, indeed, may penetrate the spot of rupture on the Graafian follicle and fertilize an ovum which has remained therein.[18-20] The spermatozoon may wander from the extremity of the tube on one side and fertilize an ovum from the opposite ovary, or an ovum of one ovary may be fertilized by a sperm cell in the opposite tube.[15]

The factors which may interfere with the passage of the fertilized ovum into the uterus are many. However, the most common cause from both clinical and pathologic evidence is some inflammatory lesion of the adnexa.

Many cases reported are not true combined pregnancy and have not been reviewed in this paper; we shall consider here only cases where the two pregnancies were concurrent and simultaneous.[16-17]

The first such case of combined pregnancy was reported by Duverney[13] in 1708, who discovered the condition at autopsy.

Since this report 290 further cases have been described. Strauss,[26] in 1898, collected thirty-two cases from the literature, while Weibel,[27] in 1905, increased the number to 119. In 1907, von Neugebauer[14] presented 169 cases, and in 1913[22] increased the number to 244. Novak,[6] in 1926, increased this number to 276 cases.

The present paper, while taking note of the articles published from 1926 to the present time, deals with only 294 cases: the 276 cases reported by Novak in 1926, together with fifteen cases since reported and three cases reported now.

The mortality was 20.7 per cent for the whole series, and 14.4 per cent if cases are excluded in which the condition was discovered only at post-mortem examination, and which did not receive treatment.

TABLE I
AGE OF MOTHER

Years	Cases
20-25	29
26-30	57
31-35	70
36-40	40
Over 41	8
Unknown	88

The maximum incidence is in the active period of childbirth, 26 to 35 years of age, with a tendency to fall in the latter half

of the period. The previous parity (Table II) suggests that previous labors predispose to the condition.

TABLE II
PARITY OF MOTHERS

Nullipara	25
Para I.	29
Para II.	45
Para III.	29
Para IV	12
Para V	10
Para VI.	8
Para VII	4
Para VIII	1
Para IX.	1
Para X.	1
Para XI.	1
Para XII.	1
Para XVIII	1
Multipara, figure not stated	8
Unknown	116

The two pregnancies may occur at the same time or the uterine conception may antedate the extra-uterine, or vice versa. A normal pregnancy may occur while the dead products of an ectopic gestation are still present.

TABLE III
DURATION OF INTRA-UTERINE PREGNANCY

Weeks	Cases
4	2
6	16
8	28
10	10
12	36
14	9
16	7
18	2
20	6
22	4
24	7
26	3
28	7
30	1
32	3
34	2
Term	88
Unknown	60

The period of pregnancy varied from eleven weeks to term. In nine cases the cause of death was internal hemorrhage. Symptoms were present from a few hours in four cases to the whole duration of pregnancy in one case. All except one of these were recorded prior to 1897.

Forty-one cases were discovered after labor had started. In this group there were six deaths. In half of this group patients showed no symptoms except an unusual increase in the size of the abdomen. In six cases the intra-uterine pregnancy was terminated by abortion.

TABLE IV
DURATION OF EXTRA-UTERINE PREGNANCY

Weeks	Cases
2	0
4	1
6	14
8	56
10	3
12	33
14	1
16	4
18	2
20	1
22	0
24	0
26	0
28	2
30	1
32	0
34	0
Term	8
Unknown	

TABLE V

	Patients Who Aborted, Weeks	Patients Whose Pregnancy Continued to Term, Weeks
Average duration of pregnancy at onset of symptoms	8.3	7.2
Average time of duration of symptoms before operation	5.2	4.7
Average time of preoperative bleeding	3 5	1.2

TABLE VI
END RESULTS

	No. of Cases	Recovered	Died
Mother.......	226	182	44
Intra-uterine fetus.......	226	103	123
Extra-uterine fetus	215	7	208

In half of the patients after delivery a symptomless mass was noted in the abdomen. The other half had varying symptoms usually suggestive of infection

or intestinal irritation. In twelve cases the symptoms were so insignificant that nothing was done for the palpable mass. Eight patients passed the fetus piecemeal through fistulae, and of this group one died. A live child was removed from the abdomen on five occasions from one to twenty-three days after labor. Sixteen patients had exploratory laparotomies, two of whom died. In the other four patients who died, there had been no operative interference. The cause of death was peritonitis in three cases and intraperitoneal hemorrhage in the fourth.

TABLE VII
DIAGNOSIS

When Made	No. of Cases	Deaths
Post-mortem........................	16	
Labor.............................	36	6
Second half of pregnancy...........	20	7
First half of pregnancy.............	219	16

In twenty patients the diagnosis was made in the second half of pregnancy or during labor. All were operated on and seven died. The causes of death were hemorrhage, septicemia, pulmonary embolism and pulmonary edema. Two deaths occurred in the sixth month, one in the seventh and four at term.

TABLE VIII
CASES DIAGNOSED IN FIRST HALF OF PREGNANCY

	Cases	Total Mortality	Treatment	Mortality
After abortion of uterine ovum.	47	7	39 laparotomies	5 due to shock and hemorrhage
			6 colpotomies	0
Before abortion of uterine ovum.	93	9	2 unoperated	2
			6 subtotal hysterectomies	1
			3 curettage	0
			83 salpingectomies	8
			1 passed fetal parts by rectum	0

CASE REPORTS

CASE I. A white multipara of 27 was admitted to Franklin Hospital July 26, 1933.

About three months before she had had a three months miscarriage, and had last menstruated May 10, 1933. Three weeks before a small amount of vaginal bleeding had occurred, accompanied by nausea and vomiting, mild abdominal pain and low backache. A week later similar bleeding occurred with pain in the shoulders and neck. A diagnosis of left tubal pregnancy or threatened abortion was made. Spotting and abdominal pain over the left tubo-ovarian region persisted.

The pulse was 82 and regular, the blood pressure 114/70, the temperature 99°F. Abdominal examination showed moderate tenderness over the left tubo-ovarian region. No rigidity was present, but muscle guarding was noted. The cervix was soft with a slight amount of blood present. The uterus was one and one-half times normal size, firm, and in anterior position. The right fornix was clear, while the left showed a small tender mass.

At exploratory laporatomy under ether anesthesia free blood was found in the pelvis. The left tube was distorted in its middle third by a mass approximately 3 cm. in diameter. This was a pregnancy which had been bleeding through the fimbriated end. The left ovary was completely degenerated. The right tube showed areas of stenosis, resulting from an old pelvic inflammatory disease. Bilateral salpingectomy and left oöphorectomy were performed. The left tube was dilated into a sac which contained a two months fetus.

The patient made an uneventful recovery and was discharged in fourteen days. Her abdomen continued to enlarge and she did not menstruate. Six and one half months later she was delivered of a normal child. (Case reported through courtesy of Drs. F. Foote and E. A. Gius.)

CASE II. A multipara of 25 was seen May 15, 1936. She complained of irregular menstrual periods and was very desirous of becoming pregnant again. Her one child, aged 4, had been delivered by cesarean section.

She was fairly well developed and in no apparent pain. Abdominal examination was essentially negative. Pelvic examination showed an hypertrophied cervix and a small uterus in first degree retroversion. The fornices were negative.

The patient was given several tampon treatments and following her period on May 23, 1936 she was insufflated. The tubes were at first found closed, but after the third insuffla-

tion the tubes opened at 110 mm. of mercury. On September 14, she again appeared, stating that her last period had been June 29, and that

FIG. 1. Preserved villi from tubal pregnancy. Case II.

she had missed the following two periods. Spotting had recurred on September 11, 1936.

The breasts were enlarged and colostrum could be expressed. The abdomen showed some tenderness to deep palpation in the left lower quadrant. The uterus was the size of a two months pregnancy and there was a small palpable mass in the left fornix, which I thought might be a cystic ovary.

The patient returned home, but spotting continued and the pain became aggravated. On September 17, she was doubled up with severe pain, bleeding intermittently. The abdomen showed muscle guarding and rigidity over the left lower quadrant. The uterus was still enlarged but a soft cystic and very tender mass was palpable in the left fornix. A diagnosis of left tubal pregnancy was made. The temperature was 99.2°F., the pulse 64, respirations 20, blood pressure 110/65.

Under gas and ether anesthesia the peritoneal cavity was opened and the old operative scar excised. The peritoneal cavity contained free fluid, but no blood. The right ovary and tube were negative. The uterus was en-

larged to about a two months pregnancy and was in anterior position. The left tube in its distal third showed a bulging dark blackish mass, an unruptured eight week tubal pregnancy. The ovary on the left side was cystic. The cysts were punctured and left salpingectomy was performed. On section, the tube showed placental tissue and a small fetus. The peritoneal cavity was closed in the usual manner.

The patient made an uneventful recovery and was discharged from the hospital fourteen days postoperatively.

The intra-uterine pregnancy developed rapidly and normally, and on March 23, 1937 the patient was delivered of twin girls by cesarean section. At this time the remaining tube was ligated.

Thus the above findings show that three eggs must have been fertilized.

CASE III. A multipara of 33, with one living child was seen by Dr. Lawrence Hoffman. She had had a miscarriage at about two and one-half months, and the flow was associated with some pain in the abdomen. This had become markedly aggravated.

A mass was found in the left cul-de-sac. An exploratory vaginal puncture was done and

blood was obtained. At laparotomy a left sided ruptured ectopic pregnancy was found and left salpingectomy was done. The patient made an uneventful recovery.

Both an extra-uterine and an intra-uterine pregnancy had been present.

SUMMARY

1. Combined pregnancy occurs with reasonable frequency, as shown by the report of 294 cases.

2. The diagnosis at times is very difficult.

3. Surgery is the treatment of choice.

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THE ACUTE ABDOMEN*

WITH SPECIAL REFERENCE TO SYMPTOMS, DIAGNOSIS AND SURGICAL TREATMENT

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THE acute abdomen is one pathologic condition encountered in the general practice of medicine in which quick thinking and sound judgment are of paramount importance, as the mortality rate, in most instances, is in direct proportion to delay. In this paper the most important and most frequently encountered pathologic conditions will be discussed.

ABDOMINAL INJURIES

Abdominal injuries, either open or closed, require immediate attention. Open injuries may be subdivided into (1) those which are limited only to the abdominal wall, and (2) those which enter the peritoneal cavity causing damage to the viscera. A stab or gunshot wound may enter the peritoneal cavity and cause no damage to the viscera but may introduce infection leading to a fatal peritonitis. For the same reason, probing of these wounds is dangerous.

The symptoms and signs of visceral injury are those of hemorrhage and shock, namely, pallor of the skin and mucous membranes, air hunger, quick, gasping respirations and sighing, cold skin with a clammy sweat, subnormal temperature, restlessness, dimness of vision, syncope and noises in the ears. The pulse is at first not affected but later becomes progressively more rapid, weak and of small volume. The blood picture will show a leucocytosis of from 19,000 to 30,000, with a diminution of the erythrocytes and low hemoglobin.

Babcock[1] differentiates shock from hemorrhage in the following manner: In shock there is a concentration of blood. There is a relative increase of hemoglobin and corpuscles, and an absence of leucocytosis. Air hunger is less marked, restlessness common and the patient more often unconscious. The patient in shock will usually show improvement when given the orthodox treatment in a reasonable length of time unless in extremis, while the patient suffering from hemorrhage will not, unless there is a spontaneous arrest or the bleeding is stopped by surgical intervention.

Closed abdominal wounds are diagnostic problems. A history of the nature of the accident is important. Symptoms of both shock and hemorrhage may be present. Physical examination reveals rigidity of the abdominal wall, hydro- or hemoperitoneum with distention and shifting dependent dulness. Pneumoperitoneum with diffuse tympany and obliteration of the liver dulness may be present. An empty leaking bladder may be found. A silent abdomen is usually diagnostic of peritonitis, whether due to blood, bile, urine or infection. The diagnosis is based upon history, symptoms and physical findings. X-ray examination is especially valuable in cases where there is a ruptured hollow viscus. The following is an illustrative case:

A young boy received a severe abdominal injury when the rear wheel of a heavily loaded truck passed over his abdomen. There was no visible external injury, but symptoms of hemorrhage were present, with painful, rigid abdomen, retraction of the knees and other signs of internal injury. Immediate laparotomy revealed about 5 inches of the mesentery torn from its attachment. The bleeding mesenteric vessels were ligated, the damaged bowel exteriorized, and the patient treated for hemorrhage and shock. Several weeks later the bowel was anastomosed. His life was saved by immediate, conservative surgery.

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The treatment of both open and closed abdominal wounds is surgical, when indicated. Open injuries involving only the abdominal wall may be treated conservatively. Meyer and Shapiro,[2] in a recent very instructive and extensive collective review, conclude in part as follows: "The policy of prompt exploration in penetrating wounds of the abdomen is established; however, no patient should be subjected to laparotomy until proper preparation has brought his systolic blood pressure above 80, better 90 or 100, unless the operation is one of 'last resort.' Secondary shock on a surgical service is equivalent to hemorrhage, and, until the bleeding vessels are ligated, should not be treated by intravenoclysis but by blood transfusion. Patients operated on in shock are less likely to survive the operative trauma. Correction of shock increases the resistance to peritonitis."

In gunshot or stab wound or in any injury to the bowel where there has been fecal contamination, the injection of 30 c.c. of coli-bactragen into the peritoneal cavity within three hours after the contamination will markedly diminish the chances of general peritonitis, and when bactragen is used it is not necessary to insert drains (Sternberg[3]).

Errors are too frequently made in attempting to do too much surgery in a patient suffering from hemorrhage or shock. Additional surgery can be performed in a second operation when the patient is a better surgical risk. It is much more logical to have a live patient with a fecal fistula or artificial anus than a dead one with a beautifully performed anastomosis.

ACUTE APPENDICITIS

Acute appendicitis is one of the first acute abdominal conditions to be considered. In spite of the advancements made in surgical diagnosis and technique in the last few years, the mortality rate in this condition has increased. Abell[4] states that there are more than 20,000 deaths per year attributable to this disease, and Herrick[5] reports that in cases with peritonitis there is an operative mortality of 5 to 30 per cent.

As emphasized by Murphy, the symptoms of a typical attack of acute appendicitis, in chronological order, are pain, nausea, tenderness and fever. The pain is colicky in nature, reaching its height in about four hours; it may start in the epigastrium or may be diffuse over the abdomen, later becoming localized over McBurney's point. Nausea, which may be followed by vomiting, appears in from two to four hours after the onset of pain. Vomiting may occur only once or twice; however, if it is continuous there is probably present either a complication or another pathologic condition. The tenderness at first is diffuse and not marked, but in four to six hours it becomes definite, localizing over McBurney's point. Rebound tenderness usually indicates peritoneal inflammation, either local or generalized, and cutaneous hyperesthesia elicited by pinching a raised fold of skin over McBurney's point is occasionally present. The fever develops in from four to twenty-four hours after the onset of pain. Usually it is not high, ranging from 99 to 102 degrees, but more frequently remaining under 100 degrees. The more acute the infection, the earlier the temperature. A sudden drop in temperature suggests gangrene, perforation or evacuation of the appendiceal contents into the cecum.

Other symptoms should also be mentioned. Constipation is usually present, although Brunn[6] states that diarrhea is not uncommon in appendicitis. Diarrhea may be present if the exciting etiologic organism is pneumococcus or streptococcus, as well as in pelvic appendicitis and many times in appendicitis in children. Tachycardia is a very important clinical diagnostic point. A pulse rate of 90 to 120 is a more reliable evidence of infection than is increased temperature. Persistence of tachycardia with remission of other symptoms suggests gangrene. Bladder symptoms may result from contact inflammation, as when the

inflamed appendix lies in proximity to the bladder or ureter.

The findings on physical examination are right rectus muscular rigidity at McBurney's point, in direct proportion to the intensity and area of the underlying peritonitis. The position of the patient aids in the diagnosis; for instance, in acute septic appendicitis the patient lies on his back with the thighs elevated and the right knee or both knees flexed. This is an important and fairly constant guide in determining the severity of the attack. Pain on contraction of the right iliopsoas muscle by raising the right knee indicates a posterior or retrocecal appendix. Vaginal or rectal examination should be done. In children rectal examination is of particular value in establishing the diagnosis. Blood count will show a leucocytosis of from 9,000 to 18,000; this usually appears early with a relatively high proportion of polymorphonuclear leucocytes. Children under 4 years of age will usually show a relative lymphocytosis.

The diagnosis of acute appendicitis is made from the symptoms and the physical findings. In my opinion the clinical findings are more important in the diagnosis of acute appendicitis than are the laboratory findings. If the leucocyte count fits the clinical picture, use it; if not, use the clinical findings alone.

The treatment of acute appendicitis is surgery, and the earlier instituted the better. If delay has occurred and if the appendix appears to be sufficiently walled off by omentum, then postpone operation as the inflammatory mass may spontaneously resolve or a definite localized abscess may develop. If the latter occurs, drain the abscess but do not try to remove the appendix as the protective wall and the abscess may be broken and a generalized peritonitis develop. The appendix should be removed later when the infection has subsided. A ruptured appendix with generalized peritonitis presents a grave problem; operation with adequate drainage probably offers the patient the best chance of recovery. Miller[7] shows that in children with generalized peritonitis the mortality rate is lowered by operation and drainage.

In the treatment of ruptured appendix with peritonitis, I have obtained extremely gratifying results by the insertion of a catheter into the ileum, if possible, or into the cecum through the appendiceal stump. The catheter is held in place by a purse-string suture around the stump and brought out through the incision or through a stab wound. Distention is relieved by compressing the bowel, ileus is prevented, and the patient's chances for recovery are greatly improved. I have never had a fecal fistula develop following this method of treatment.

Decompression of the cecum as described above has been advocated by DeCourcy,[8] who shows a reduction in mortality to 4 per cent in a series of fifty cases.

Serum therapy has been used by Priestley and McCormack[9] in the treatment of generalized peritonitis secondary to rupture of the appendix with some hope of reducing the mortality from this complication.

INTESTINAL OBSTRUCTION

Mechanical intestinal obstruction is the type of ileus which requires immediate surgery and, as in other acute abdominal conditions, the quicker the patient is submitted to surgery the better are the chances for recovery.

Intestinal obstruction may be caused by:

1. Pathology without the bowel, strangulated hernia either internal or external being the most common cause. Adhesive bands, either congenital or postoperative, are also frequently causative factors. Other causes are tumors (renal, splenic, pancreatic, ovarian and mesenteric), lymphatic inflammation, new growths, uterine and ectopic pregnancies, and occasionally gall- or urinary bladder stones.

2. Pathology within the bowel is caused from impacted feces, foreign bodies, parasites, hair balls, and gallstones which if

large enough will cause obstruction at the ileocecal junction.

3. Pathology developing from the wall of the bowel, such as tumor or carcinoma, is a common cause of intestinal obstruction. Carcinoma may be the cause of a chronic or partial obstruction and then may suddenly close the lumen, causing an acute obstruction. This is to be considered especially in patients within the cancer age group who give a history of abdominal distress with a change in bowel habit. The higher the obstruction the more severe will be the symptoms.

In children intussusception is the most common cause of obstruction. It is diagnosed by abdominal pain, early vomiting, palpable mass upon rectal examination with the characteristic "cranberry sauce" blood on the examining finger, associated with abdominal distention. Christopher[10] has recently reported two additional cases of intussusception in adults, one a high jejunal intussusception caused by a papillary adenoma and the other a chronic ileocecal intussusception.

Acute obstruction is a complication of regional enteritis, a recently described pathologic condition about which there is much discussion at the present time. Jackson[11] has reviewed the subject and reported several additional cases.

Other pathologic conditions, developing from the wall of the bowel and causing obstruction are strictures from healed ulcers, tuberculous and syphilitic lesions, chronic enteritis, diverticulitis and peri-diverticulitis chiefly of the sigmoid and pelvic colons, benign tumors of the bowel, such as adenomata, lipomata, myomata, polyposis, endometrial adenomata and hemangioma. Mucocele of the appendix or hematoma of an appendix epiploica, while very rare, may give rise to acute obstruction.

The symptoms of intestinal obstruction are usually constant and include vomiting, projectile in type and containing first the contents of the stomach, then bile, and then intestinal contents. The loss of hydrochloric acid in the stomach contents leads to alkalosis, while the loss of pancreatic juice and bile leads to acidosis. The pain of the abdominal colic is centered around the umbilicus and is of a twisting, cutting, griping nature. It is constant with exacerbations if the obstruction is complete, while with incomplete obstruction there are remissions of the pain, and if gangrene is present there is temporary cessation of vomiting and pain. Meteorism or gaseous distention of the intestine is present except in high obstruction. No abdominal rigidity is present except when there is, an associated peritoneal irritation. A noisy abdomen with a metallic tinkling and splashing sound is characteristic in the early stages. Obstipation is a constant finding; the feces in the bowel beyond the obstruction may be passed but little or no gas will be expelled.

The diagnosis of acute mechanical obstruction is made by the presence of intermittent, crampy, colicky abdominal pain followed by nausea and projectile fecal vomiting, without tenderness, rigidity or fever. X-ray examination either by flat plate or by fluoroscopy may reveal step ladder-like arrangement of distended small bowel, especially if the obstruction is low. In acute cases, barium is not used in the x-ray examination.

The treatment of acute intestinal obstruction is surgical removal of the cause of obstruction as soon as possible after the diagnosis is made and with as little operative trauma and shock as possible. Prolonged and extensive operations are condemned. If a gangrenous piece of bowel is found, exteriorize it and do an enterostomy or colostomy. Later, when the patient is in better physical condition, the bowel can be anastomosed. When the viability of a piece of bowel is questionable, this piece may be brought up just beneath the skin surface in the incision and packed away from the abdominal cavity with gauze so as not to interfere in any way with the blood supply. If the viability of the bowel returns, nothing more need be done except to remove the

gauze and allow the wound to close. If, however, the questionable piece of bowel becomes necrotic, an enterostomy will spontaneously occur and, thereby aid in saving the life of the patient. Ileostomy, colostomy or cecostomy may be performed proximal to the obstruction; these procedures decompress the bowel and allow it to assume its normal size and function before any further surgery is attempted. Anastomosis should never be attempted in a piece of bowel which is markedly distended.

Sufficient fluids in the form of normal saline with or without glucose should be administered by venoclysis or hypodermoclysis to restore the lost chlorides. Preoperative administration is equally important with postoperative. The amount varies with the individual case and with the condition of the patient, but usually from 2,000 to 3,000 c.c. in twenty-four hours is sufficient. A Levine tube in the stomach with suction may be necessary to relieve the distention. Narcotics postoperatively may be used when necessary.

PEPTIC ULCER

The term "peptic ulcer" includes both duodenal and gastric ulcer. A serious complication in this condition is hemorrhage which is usually treated medically. Allen[12] states that duodenal ulcers rarely cause fatal hemorrhage under the age of 50 years, while beyond this age the mortality is 33⅓ per cent. He advises the operative procedure of ligation of the artery supplying the ulcer in the better risk older group cases. Means[13] believes that operation should be limited to patients bleeding after 50 years of age or those having repeated hemorrhage.

Acute perforation occurs in a certain percentage of chronic ulcers. The symptoms are violent, acute abdominal pain which may cause the patient to fall to the floor or be unable to move. This pain may be referred to the epigastrium, right abdomen, scapula or back. Shock with pallid face, sweating brow, staring eyes may be present. The patient appears to be in agony and very apprehensive. Board-like abdominal rigidity is present, which is more marked over the ulcer or McBurney's point with the patient in a flexed position. These patients cannot lie flat; to avoid pain they lie in a flexed position. Breathing is shallow, jerky and costal. Temperature is normal or slightly subnormal. Pulse is slow, full and of normal volume until peritonitis develops. Vomiting is absent unless fluid or food is taken.

Physical examination reveals free fluid, rarely gas in the abdomen, obliteration of liver dulness, shifting dulness in the flanks and evidence of early peritonitis in the epigastrium and right iliac fossa. Vomiting of blood or its presence in the peritoneal cavity is unusual.

In acute perforation of an ulcer the diagnosis is made by the history of sudden and very violent epigastric pain, causing apparent shock, board-like rigidity of the muscles of the anterior abdominal wall, tenderness and dulness in the flanks and pelvis. Pneumoperitoneum may be present. There may be cessation or absence of abdominal peristalsis. The condition occurs most frequently in males who may or may not give an ulcer history. Flat x-ray plate or fluoroscopic examination usually shows air under the diaphragm and this is considered quite diagnostic.

The treatment of perforated peptic ulcer is immediate laparotomy to stop the leak. Any lapse of time after six hours from the time of perforation gives a poor prognosis, and after thirty-six hours the majority of patients die. Since these ulcers usually rupture on the anterior wall, the location of the perforation is quite evident, but because they generally are in shock and are poor surgical risks, the least amount of surgery done is good judgment, unless there are other indications. At operation the perforation is closed with a purse-string or superimposed Lembert suture. A piece of omental fat is attached over the sutured perforation and the abdomen is closed. A gastroenterostomy or any other

additional gastric surgery at this time is to be condemned, unless there is marked pyloric or duodenal obstruction. It is better judgment to perform any further gastric surgery, if necessary, at a later date when the patient is in better condition. The question of drains is controversial. Since the gastric contents are primarily sterile, many surgeons close the abdomen without drainage; others use drains and remove them early.

Postoperative fluids by venoclysis are essential. These patients should receive medical treatment for the ulcer following operation.

RUPTURED ECTOPIC PREGNANCY

These patients present symptoms of early pregnancy and rupture usually occurs before the twelfth week. The prodromal symptoms are sharp, cutting, crampy, low abdominal pains progressing in severity as the pregnancy advances. Abnormal vaginal bleeding or spotting of blood is present in most cases. Decidual tissue may be passed, suggesting an incomplete abortion. Weak spells, sometimes severe enough to cause fainting, may be experienced. When rupture occurs there is sudden, severe abdominal pain, shock, collapse and usually evidence of intraperitoneal hemorrhage; the pulse rate is increased and of small volume; temperature is subnormal early but later may be elevated; vomiting sometimes occurs.

The Aschheim-Zondek or Friedman test will often determine the diagnosis before rupture or tubal abortion occurs. Bimanual examination reveals a soft and sometimes enlarged uterus. Marked tenderness in the pelvis over the affected adnexa and a palpable mass may be felt. The abdomen is tender and usually without rigidity. The cul-de-sac feels full and doughy. Posterior colpotomy with recovery of blood often aids in making a diagnosis. One can determine the presence of free blood in the peritoneal cavity by puncturing the posterior cul-de-sac with a large needle and aspirating its contents. Hope[14] has shown the value of the peritoneoscope in the differential diagnosis of this condition, and Ruddock[15] shows its value in the diagnosis of other abdominal and pelvic pathology.

Severe hemorrhage from a ruptured graafian follicle gives a similar clinical picture, except that the symptoms of pregnancy are lacking. The treatment, however, is usually the same as for ruptured ectopic pregnancy.

As soon as the diagnosis is established, immediate operation should be performed to stop the hemorrhage. Shock and depression may be out of proportion to the loss of blood and in those cases a delayed operation might be advisable until the patient is in better condition. This, however, is a point difficult to decide clinically. In most cases this condition should be considered as a surgical emergency. Evidence of intraperitoneal hemorrhage should indicate laparotomy. Removal of the ruptured tube and ligation of all bleeding points should be done as soon as possible, with immediate closure. Removal of the opposite tube or the appendix should not be done, except in the presence of gross pathologic changes when the condition of the patient permits further operation.

Blood transfusions are especially valuable in these cases and should be given after the bleeding is stopped. Shock is treated by venoclysis and hypodermoclysis along with the blood transfusions, and general supportive treatment should be instituted.

ACUTE PELVIC CONDITIONS

Twisted ovarian cysts and uterine fibroids with impending gangrene are other pathologic pelvic conditions which require immediate surgery. Acute salpingitis is best treated conservatively.

ACUTE CHOLECYSTITIS

The etiologic factors responsible for acute cholecystitis are (1) gallstones impacted in the cystic duct which interfere with emptying of the gall-bladder and at times produce, as a result of pressure,

interference with the circulation with resultant edema, congestion and occasionally gangrene and perforation; (2) hematogenous conditions, as from bacteria circulating in the blood stream during or following typhoid fever, septicemia, pneumonia and influenza. Rosenow's experiments led him to believe that streptococci in the blood stream have a peculiar elective affinity for the gall-bladder. (3) Bile infection from bacteria in the alimentary tract. Typhoid fever in earlier life has always been associated with this mode of infection. Potter[16] believes that biliary diseases in children may be caused by contributary factors, such as upper respiratory infections, including influenza, pneumonia, scarlet fever, appendicitis, intestinal parasites and sometimes a history of abdominal trauma. (4) Other theories suggested as a mode of infection are lymphatic-borne infections, infections due to propinquity from another organ, ascending biliary infections and amebiasis.

Acute cholecystitis is grouped pathologically by Taylor[17] into (1) acute edematous, (2) acute suppurative, and (3) acute gangrenous gall-bladder, which may be further subdivided into acute gangrenous infarct and acute suppurative gangrene. This grouping is based on the pathologic findings in the gall-bladder wall.

Symptoms of acute cholecystitis depend upon whether or not the cystic duct is obstructed by a stone. In the simple non-obstructive type the attack may be very slight and commonly is called acute indigestion or acute biliousness, rarely requires morphine for relief, and usually subsides under medical management. Acute obstructive cholecystitis is characterized by a sudden, severe attack of pain in the upper right abdominal quadrant or in the epigastrium and may radiate to the back in the region of the right eleventh rib, or to the right scapula. This pain is due to the passage of a stone down the cystic duct or its impaction in the neck of the gall-bladder. The pain is accentuated by the slightest diaphragmatic movement. Relief

may be obtained by attempting a new position, as bending, putting pressure over the back, or by taking a hot bath. The intensity of the pain may become so great as to require morphine. Vomiting and constipation are almost always constant accompaniments. Vomiting may bring relief and is many times induced by the patient. Chills may be severe but the fever rarely rises above 102 degrees. The severity of the infection is indicated by the temperature. Marked rigidity over the right rectus muscle and extreme tenderness on pressure over the right upper quadrant are present. A palpable gall-bladder which moves with respiration may be found, but only if rigidity of the right rectus muscle is slight or absent. Jaundice is rare in uncomplicated cases.

The history of previous dyspepsia or distaste for certain foods, such as fats, with the above symptoms, is diagnostic of acute cholecystitis. The statistics of Mentzer[18] show that 72 per cent of the cases are in females. Leucocytosis of a moderate degree is usually present but many times is misleading as to the true underlying pathology and cannot be relied upon too much. X-ray by means of the gall-bladder dye or flat plate may be of value.

The treatment of acute obstructive cholecystitis is surgical, whether it be removal of the gall-bladder, drainage, or a combination of the two. In my opinion, all these methods have their merits in especially indicated cases and no set rule can be established. However, Puestow,[19] in recent experimental work, showed that removal of the gall-bladder causes a dilatation or paralysis of the sphincter of Oddi which allows free drainage from the bile passage, thereby reducing intraductal pressure. From this it appears logical that cholecystectomy should take precedence over the other methods. The present tendency is toward early cholecystectomy, as shown by the writings of Heyd,[20] Clute,[21,22] Mentzer,[18] Heuer,[23] and others. They believe that cystic duct obstruction is the first event in the pathologic process

with infection secondary to the obstruction. They therefore advocate removal before the secondary infection occurs.

Preoperative administration of glucose and saline should be given, preferably by venoclysis. This stores glycogen in the liver, corrects any abnormal chemistry in the body, and balances the body fluids. Postoperative care must include adequate fluid intake and the treatment of complications, if any occur.

ACUTE PANCREATITIS

While this condition is rather infrequent, it is one of the most serious and carries one of the highest mortality rates of all acute abdominal conditions. There are several theories as to its etiology (Lewis[24]) but the "common channel theory" is the one most commonly accepted. Since it usually follows or is associated with cholelithiasis, it is thought that the ampulla of Vater becomes occluded or partially so, causing bile to flow from the common duct into the duct of Wirsung. The anatomic relations of these two ducts could in the majority of cases make this possible.

The pathology is a matter of the degree of destruction, ranging from acute hemorrhagic pancreatitis to acute gangrenous pancreatitis to acute suppurative pancreatitis, which may be diffuse or local.

The *onset* of symptoms is sudden and violent with agonizing, excruciating pain. The pain is so severe that large repeated doses of morphine may be required. Shock is a characteristic finding and in very severe cases is accompanied by cyanosis, pallor, cold and clammy skin. The temperature is subnormal with a rapid, weak pulse. Vomiting of bile and stomach contents usually occurs, and jaundice may appear early. The abdomen is distended and gives evidence of free fluid and early peritonitis. Epigastric tenderness and rigidity in the left upper quadrant is present.

Diagnosis is difficult, but the history of gall-bladder disease and sudden onset with shock are helpful along with an increase usually of the blood amylase.

DeKlimko[25] values the leucocyte count highly; it usually varies from 12,000 to 20,000. The symptoms closely simulate those of acute perforated peptic ulcer and acute cholecystitis and later bowel obstruction.

The treatment of acute pancreatitis is surgical. This consists usually of a right transrectus vertical incision. The purpose is to evacuate pancreatic fluid, clots, necrotic masses and to decrease the pressure of bile in the ampulla of Vater. Abell[26] states that the pancreatic tissue should not be interfered with but that gauze or drains should be inserted down to it. If the condition of the patient permits, the bile tract may be drained. This is especially indicated in the more chronic cases. Recently discussion has arisen as to the advantage of delayed operation. This is probably advisable in selected cases in the hands of selected surgeons, but due to the difficulty of correctly diagnosing the condition there probably would be many ruptured peptic ulcers diagnosed as acute pancreatitis by the average surgeon and hence delay would, in the majority of cases, mean the death of the patient.

DIVERTICULITIS

Diverticulitis is inflammation of a preëxisting intestinal diverticulum. This diverticulum may be present in any part of the gastrointestinal tract and give no symptoms until complications arise. One frequently encounters Meckel's diverticulum located in the lower ileum about 30 to 150 cm. above the ileocecal valve. Occasionally it is lined, or at least partially so, by tissue which histologically is the same as gastric mucosa containing chief and acid cells secreting hydrochloric acid. Ulceration and perforation may occur in these cases.

Symptoms may be lacking, but when present, are due to complications, as catarrhal inflammation (Meckel's diverticulitis), ulcerations, hemorrhage, perforation or gangrene. In such instances the symptoms consist of pain, nausea, vomiting, localized tenderness, rigidity and in-

creased leucocyte count. These symptoms make it almost impossible to differentiate it from appendicitis. Other conditions which require differentiation are strangulation of the bowel as in a hernial sac, twists or attachments causing symptoms of bowel obstruction, and foreign bodies retained within the lumen.

The treatment for inflammation or obstruction is the operative removal of the diverticulum with closure as in appendicitis. Adherent or obstructed intestine should be freed or, if gangrenous, resected or exteriorized with an enteroanastomosis or enterostomy.

The colon, and especially the sigmoid, are the most frequent locations for herniations of the mucosa through weakened circular muscle. These herniations often enter the fatty tabs, cause rotation of the bowel toward the mesenteric attachment, collect and retain fecal masses, cause irritation, ulceration, and many times perforation. Carcinoma occasionally results.

Symptoms are chronic constipation, chronic catarrhal colitis with passage of blood-stained mucus. In acute inflammation there is sudden acute pain in the left lower quadrant or in the pelvis, with lower abdominal rigidity and sometimes a palpable tender mass, with fever and leucocytosis. Perforation may occur, usually into the bladder. X-ray and proctoscopic examination aid in the diagnosis, as does the history of an obese person over 50 years of age with chronic constipation.

Treatment is medical in about 85 per cent of cases, if there are no complications. Surgical treatment of the complications is often only palliative.

MESENTERIC THROMBOSIS

Mesenteric thrombosis is fortunately an infrequent disease, attacking chiefly men past 40 years of age, usually with a history of myocarditis, endocarditis, or arteriosclerosis. The superior mesenteric artery is most frequently involved, rarely the inferior mesenteric, as shown by Gambee.[27]

Symptoms are sudden, violent, diffuse abdominal pain with collapse, followed by hematemesis and melena. Temperature is subnormal early but later rises with signs of ileus and peritonitis.

Diagnosis is extremely difficult and is rarely made preoperatively. If the patient has had previous heart disease or arteriosclerosis with the above symptoms, if there is a palpable mass, a leucocyte count of 20,000 or higher, with 85 per cent or more polymorphonuclear leukocytes, the diagnosis is more certain. Acute pancreatitis, perforated peptic ulcer and intestinal obstruction must be differentiated.

Treatment is immediate laparotomy, the necrotic bowel being exteriorized or resected. The entire intestinal tract should be carefully inspected so that no infarcts are overlooked.

GENERAL CONDITIONS

The following are some conditions which may simulate an acute abdomen but do not require abdominal surgery: Renal colic, especially if on the right side, may closely simulate acute appendicitis; pneumonia and acute pericarditis are common causes of abdominal pain in children; food and lead poisoning may become confused with an acute surgical abdomen; tabetic crisis often simulates an acute abdomen in the adult; coronary disease, thrombosis, angina pectoris (Mohler,[28] Morrison,[29] Anderson,[30] Patterson,[31] Burns,[32] and Aynesworth[33]); spontaneous idiopathic hemopneumothorax (Hurxthal[34]); mesenteric lymphadenitis (Bell[35]); pulmonary tuberculosis (McReynolds[36]); juvenile diabetes mellitus (Newcomb[37]); blood dyscrasias, as Henoch's purpura, sickle-cell anemia and abdominal allergy (Althausen, Deamer and Kerr,[38] Campbell,[39] Davis[40]); subarachnoid hemorrhage (Thurston[41]); hemorrhage into a hydatid of Morgagni (Zener[42]); arachnoidism (Morton[43]) and periarteritis nodosum (Sawyer[44]).

SUMMARY

1. Pain is the chief symptom of the acute abdomen.

2. Early diagnosis of an acute surgical abdomen is essential.

3. The most frequent and important surgical conditions of the abdomen have been discussed.

4. Diseases both common and rare which give abdominal pain but do not require abdominal surgery have been mentioned.

5. Immediate conservative surgery, when indicated, has been stressed.

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PEPTIC ULCER OF MECKEL'S DIVERTICULUM

CASE REPORT AND REVIEW OF THE LITERATURE

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ALTHOUGH peptic ulcer of Meckel's diverticulum, giving rise to its various late symptoms, has been known to the pathologist for almost a century, it has been only during the last decade that clinicians have attempted to develop a definite symptom-complex that would enable them to diagnose preoperatively a condition which, judging from published reports of the past few years, is by no means as rare as the textbooks would have us believe.

Uncomplicated Meckel's diverticulum has been described as occurring in 1.5 to 3 per cent of all persons, being present, according to Harbin,[27] from 14 to 16 inches (35 to 40 cm.) from the cecum. Normally the vitelline duct undergoes complete obliteration during the seventh week, but the duct, or segments of it, may persist. When the remnant of the primitive duct proximal to the ileum persists, it forms what is called Meckel's diverticulum. Heterotopic tissue is present in approximately 25 per cent of all Meckel's diverticuli.

Numerous theories have been proposed to explain the presence of such aberrant elements as gastric and duodenal mucosa and even pancreatic tissue "rests." The earliest and most commonly accepted theory is that of Albrecht[3] which maintains that the entoderm lining the primitive intestinal tube possesses the potentiality of developing into any of the glandular components of the fully developed gastrointestinal tract. Shaetz[56] proposes that during early embryonic life there may be a transference of endodermal cells lining the primitive intestinal tube, due to the rotating movements of the embryo, with re-implantation of these cells at narrowed points in the intestinal tract. Farr and Penke[18] hold that the vitello-intestinal duct originally may have had a digestive function and hence embodied a complete primitive digestive system.

Greenblatt, Pund and Chaney[23] add the theory of "dysembryoma" in which they agree with Albrecht that the entodermal lining does form cell groups which function as a distinct primitive digestive system such as Farr and Penke suggest. They go one step farther in stating that while normally this embryonic system retrogresses as soon as its function ceases, occasionally "a vestige of heterotopic tissue remains as a consequence of retarded embryological retrogression of the omphalomesenteric duct." Certainly the nutritive elements within the early yolk sac are assimilated by the embryo and we are forced to agree that these elements certainly must be acted upon by the cells of the vitelline duct in some manner analogous to passage of food through the upper digestive tract before it is absorbed by the cells of the jejunum and ileum.

When present, the heterotopic tissue usually lines the greater portion of the diverticulum, often involving the neck of the sac. Hence surgical removal should not consist of merely excising the anomaly. A small length of ileum extending to each side of the diverticulum should be removed, and anastomosis, preferably lateral, performed; otherwise we cannot be sure that all of the gastric or duodenal cells, if present, have been removed. The patient then runs the risk of later developing a peptic ulcer of the ileum with a repetition of his original symptoms.

Greenblatt and his associates emphasize the difficulty of correct diagnosis because of the possible various symptom combinations which may so closely imitate one of several different and more common acute abdominal conditions. They have analyzed a series of eighteen cases of Meckel's diverticulum observed during the past ten years at the University Hospital of the University of Georgia School of Medicine. They divide these into six groups. (Table I.)

Meckel's diverticulum with peptic ulcer, can be made, as we will attempt to show. The cardinal symptoms and signs are: (1) history of massive intestinal hemorrhage; (2) an average age of from 10 to 15 years; and (3) abdominal pain.

Practically all writers agree that the cardinal symptom of peptic ulcer of a Meckel's diverticulum is hemorrhage. It was present in twenty-nine of the thirty-four cases reported by Greenwald and

TABLE I

Group	Findings		Symptomatology
Peptic....................	Gastric mucosa	without ulceration with ulceration ulcer and hemorrhage ulcer and perforation	May simulate duodenal ulcer. History of intestinal hemorrhage. Peritonitis due to perforation.
Obstructive...............	Intussusception Volvulus Bands and adhesions Contents of inguinal or femoral hernia		Signs and symptoms of intestinal obstruction may vary from chronic to acute; partial to complete obstruction.
Diverticular...............	Simple acute Acute with perforation and gangrene Chronic		Symptomatology essentially that of appendicitis.
Umbilical.................	Fecal fistula Umbilical adenoma Prolapse of intestine through umbilical fistula		Lesions of umbilicus often associated with underlying omphalomesenteric duct.
Tumor....................	Benign...............	enterocystoma carcinoid adenoma mesodermal tumors	Symptoms of bleeding, intussusception or obstruction.
	Malignant...........	carcinoma sarcoma	
	Heterotopic.........	pancreatic tissue embryonal rests	
Incidental.................	Normal intestinal structure		None

Clinically the diagnosis of groups two, three, five and, naturally, six is practically impossible because of the absence of any clear cut differential sign that might rule out the more frequently encountered abdominal lesions. The diagnosis of group four becomes possible because the presence of tumors, fistulas or cysts of the umbilicus would suggest the presence of a patent omphalomesenteric duct within the abdomen. The diagnosis of the lesions in group one,

Steiner.[24] In five of these cases it was the only symptom and extended over a period of from one day to nine months. In a second group of five cases the initial symptom was intestinal hemorrhage which was subsequently followed by abdominal pain. The hemorrhages were of longer duration in this group and were present for from two months to four years. In the third group of five cases the onset occurred with abdominal pain but with hemorrhages appearing

later. In this group the course was more acute, the duration of the bleeding being given as from twenty-four hours to two weeks. Of the group of seventy-seven histologically proved cases in Chart 1 sixteen, or approximately 20 per cent, contain no mention of intestinal hemorrhage having been present. An analysis of these cases (Table 11) reveals the interesting

TABLE II

ANALYSIS OF CASES IN WHICH NO HEMORRHAGE WAS REPORTED

Case No.	Preoperative Diagnosis	Gross Findings
1	Perforated appendix	Perforated ulcer at base; peritonitis.
4	Appendicitis	Perforated ulcer at edge of gastric mucosa.
15	Appendicitis; peritonitis	Perforated ulcer partly walled off. Local peritonitis
21	Perforated appendix	Perforated ulcer at base with peritonitis.
23	?	Umbilical cyst containing gastric juice. Ulcer at base.
24	Peritonitis	Perforated peptic ulcer. Peritonitis.
26	Peritonitis	Two ulcers at neck; one perforated.
29	Perforated appendix	Perforated ulcer at base with peritonitis.
35	Peritonitis	Perforated ulcer at base found at autopsy; peritonitis.
37	?	Chronic ulcer at neck of diverticulum.
40	Acute appendicitis	Perforated ulcer. Walled off.
42	Peritonitis, appendiceal	Adherent mass. Lentil shape base sealed to abdominal wall.
43	?	Chronic peptic ulcer at base of diverticulum.
47	Acute appendicitis	Lentil shape perforation at base sealed to abdominal wall.
58	Duodenal ulcer with adhesions	Superficial erosion of mucosa of diverticulum.
71	Acute appendicitis	Perforated ulcer of base of diverticulum.

observation that in all but three cases the ulcers *had already perforated*, causing either local or diffuse peritonitis. Of the three remaining unperforated cases, two (Case 23

and Case 58) would, perhaps, not belong to the peptic group of Meckel's diverticulum according to Greenblatt's classification. It would be interesting to review the history of each of these cases now that the emergency of an acute abdomen has passed; perhaps the examiner's questioning, no longer prejudiced by the patient's appearance, temperature and rigid abdomen, would have taken a wider view and elicited the fact that hemorrhage *had* been present at some time or another prior to hospitalization of the patient.

Usually the patient gives a history of previous massive intestinal hemorrhages followed by a relatively long period—months and even years—before recurrence. The hemorrhage is usually massive although in some cases only slightly bloody stools have been seen. The stool is composed of bright red to black blood and may be fluid or semi-clotted. It is not mixed with mucus as in the dysenteries, nor does it have the raspberry jam appearance of intussusception. Alarmingly rapid exsanguination to the point of collapse indicates the seriousness of the condition. Moll[50] reported a case in which the bleeding began two days after birth and continued intermittently up to five months when laparotomy was performed. Callender[10] reported the case of an infant who died thirty-six hours after the first hemorrhage. However, hemorrhage may persist over a long period of time. Megevaud and Dunant[47] cite cases in which hemorrhage had occurred intermittently from childhood to adult life.

Conditions besides *intussusception* and the *dysenteries* which may simulate bleeding peptic ulcer of a Meckel's diverticulum are: *Henoch's purpura* which may be ruled out by the absence of purpuric spots, normal bleeding and clotting times, normal clot retraction and normal platelet count; *bleeding peptic ulcer* is somewhat more difficult to eliminate because some cases of diverticular ulcer do have a definite pain-food relation. However, the pain of duodenal ulcer is usually epigastric in position, there is a more prompt response to the

effects of the alkalis, and the duodenal ulcer will usually be revealed by x-ray study. *Rectal polyp* may bleed profusely but a history of tenesmus and the aid of the proctoscope may prove the absence of this lesion. The x-ray may or may not be of value in outlining the mass. *Ruptured rectal varicosities* may cause sudden massive hemorrhage similar to the bleeding diverticular ulcer, but here again the proctoscope affords a means of differentiation. *Congenital intestinal telangiectasis* is so rare that it is mentioned only to be dismissed as mathematically improbable. It is our opinion that, regardless of other symptoms, a history of massive hemorrhage from the rectum followed by recovery and then recurrence of the hemorrhage after the lapse of weeks or months is almost pathognomonic of peptic ulcer of a Meckel's diverticulum.

TABLE III

AGE INCIDENCE OF PEPTIC ULCER OF MECKEL'S DIVERTICULUM

Age	No. of Cases	Percentage
1 or less.	8	10.8
1 to 10.	34	45.9
11 to 20.	21	28.3
21 to 30.	9	12.1
31 to 40. .	0	0
41 to 50. .	2	2.7
Age not reported	2	2.7

Peptic ulcer of a Meckel's diverticulum is an affection of childhood. The first and second decades of life are most frequently involved although no age group is immune. Kleinschmidt[37] reported a case in a patient 45 years old, and McKeen had a patient 53 years old in whom this condition was associated with duodenal pathology. In the series of cases summarized in this paper approximately 75 per cent were found in patients less than 20 years old, with the greatest number of cases occurring in the age group of from 1 to 10 years. (Table III.)

Johnston and Renner's[36] excellent review, which we have taken the liberty to use as the basis of our compilation of cases, shows that the ulcers were discovered principally in infants and children: 14 per cent were less than 1 year old; 18 per cent were between 1 and 2 years; 5 per cent between 3 and 4; 23 per cent were 5 to 10 years of age; 24 per cent from 11 to 16; and the remainder from 16 to 53 years of age. The age was not given in two cases, although Treplin's case is reported as being a child.

Pain is present in a majority of the cases but may vary from mild abdominal distress on to the agonizing paroxysms of peritonitis. Of course the extreme degree of abdominal pain only follows perforation of the anomalous viscus. This symptom would be rendered obsolete if the surgeon recognized the underlying pathologic condition in its unruptured stage. The most frequent type of pain encountered is a colicky cramp-like sensation referred to the umbilical or hypogastric regions, and is probably due to distention of the small bowel or colon by the enormous amount of blood and clots within their lumen; however, several cases reported indicate a definite relationship of pain to food, just as in duodenal ulcer. Kleinschmidt's patient developed pain regularly one and a half hours after meals; Megevaud and Dunant's patient was relieved by food, and Greenblatt et al. report in their series the case of a man 47 years old whose pain was regularly relieved by doses of sal hepatica. The pain, regardless of degree, may precede or follow the hemorrhage and usually bears no relationship to meals.

Tenderness and rigidity are absent and palpation may either cause a moderate amount of discomfort or may be negative. Perforation of the ulcer, of course, will yield a picture of peritonitis and an etiologic diagnosis, except possibly from the history, becomes impossible. Pain, therefore, while a valuable aid to the diagnosis of peptic ulcer of Meckel's diverticulum, if it occurs in the classical manner, may manifest itself in so many possible degrees and characterizations that

CHART I

REVIEW OF HISTOLOGICALLY PROVED CASES

Author	Year	Age	Sex	Pain	Intestinal Hemorrhage	Acute Abdomen	Preoperative Diagnosis	Gross Findings	Histologic Findings	Result	Remarks
1. Deetz[13]	1907	9	M	+	−	+	Perforated appendix	Perforated ulcer at base Peritonitis	Gastric and pancreatic mucosa	Recovered	First definite clinical case
2. Hubschmann[31]	1913	4½	M	+	+	+	Peritonitis	Perforated marginal ulcer Peritonitis	Ulcer at edge of gastric mucosa	Died	
3. Callender[10]	1915	10 mo		−	+	−	Autopsy Death of hemorrhage Appendicitis	Punched out ulcer at base, eroded vessel	Gastric and ileac mucosa	Died	
4. Gramen[22]	1915	10	M	+	−	+	Appendicitis	Perforated ulcer Peritonitis	Ulcer at edge of gastric mucosa	Recovered	
5. Meulengracht[40]	1918	12	M	+	+	−	Autopsy Death of other cause	Ulcer about to perforate	Ulcer at edge of gastric mucosa	Died	
6. Mueller[51]	1919	11	M	+	+	+	Peritonitis	Perforated ulcer	Ulcer at edge of gastric mucosa	Recovered	
7. Megevaud and Durant[17]	1922	28	M	+	+	−	Duodenal or intestinal ulcer	Ulcer at base of diverticulum	Ulcer at junction of gastric and ileac mucosa	Recovered	
8. Schreuder[53]	1923	8	M	+	+	−	Duodenal ulcer (x-ray).	Thickened pylorus Ulcer at base of diverticulum	Pancreatic tissue at tip	Recovered	
9. Brusser[9]	1924	15	M	+	+	+	Tumor or polyp Autopsy	Perforated ulcer at base Peritonitis	Ulcer at junction of gastric and ileac mucosa	Died	Perforated 8 days after exploration for tumor or polyp.
10. Guibal[25]	1924	14	M	−	+	−	Intestinal tuberculosis	Chronic penetrating marginal ulcer Eroded vessel	Ulcer at junction of gastric and ileac mucosa	Recovered	
11. Humbert[34]	1924	11 mo	M	+	+	+	Peritonitis	Perforated ulcer. Peritonitis	Ulcer at junction of gastric and ileac mucosa	Died	
12. Pisculc[52]	1925	41	M	+	+	−	Ulcus simplex	Ulcer at Meckel's diverticulum	Peptic element confirmed	Recovered	
13. Ulrich[62]	1925	8 mo	M	−	+	−	Peritonitis	Perforated ulcer Peritonitis Blood in peritoneal cavity	Bleeding ulcer at base, gastric mucosa present.	Died	
14. Abt and Strauss Case number 1	1926	2	L	+	+	−	?	Ulcerated and inflamed diverticulum	Gastric mucosa at apex; infiltrated and fibrotic as around peptic ulcer.	Recovered	
15. Etchegorry[17]	1926	16	M	+	−	+	Appendicitis, peritonitis	Perforated ulcer partly walled off	Gastric mucosa distal to ulcer of diverticulum.	Recovered	
16. Klenschmidt[37]	1927	15	M	+	+	+	Appendicitis	Perforated ulcer at base Peritonitis	Gastric mucosa present.	Recovered	Perforated while under observation
17. McCill[10]	1927	4	M	+	+	+	Autopsy	Perforation at base Abscessed abdominal wall Peritonitis	Ulcer at junction of gastric and ileac mucosa.	Died	Perforated while under observation.
18. Jackson, A S[35]	1927	14	M	+	+	+	Ulcer of Meckel's diverticulum Autopsy	No gross ulcer	Gastric mucosa and area of ulceration	Recovered	First case diagnosed clinically
19. Taylor[58]	1927	17 mo	M	−	+	−	Autopsy	Intramesenteric tumor with overlying chronic ileac ulcer	Ulcer bordering a zone of gastric mucosa	Died	Exsanguination
20. Meiss[48]	1928	2	M	−	+	−	Intussusception or polyp	Chronic ulcer at tip, perforating ileum at a higher level Ileum full of blood.	Gastric and ileac mucosa	Recovered	
21. Hartglass[28]	1928	14	F	+	−	+	Perforated appendix.	Perforated ulcer at base Peritonitis	Ulcer at junction of gastric and ileac mucosa	Recovered	
22. Peterman and Seeger[53]	1928	6	M	+	+	+	Ulcer of Meckel's diverticulum	Perforated chronic marginal ulcer; ulcer at tip perforating ileum at higher level, also two ileac ulcers.	Gastric mucosa present.	Recovered	At first operation two weeks previously blood in peritoneal cavity.
23. Treplin[61]	1929	child	?	−	−	+	Previous appendectomy; second operation for recurrent pain.	Umbilical cyst containing gastric juice Peptic ulcer at neck of diverticulum	Ulcer in ileac mucosa; islets of gastric mucosa.	Recovered	

No. and Author	Year	Age	Sex					Clinical Diagnosis	Operative Findings	Pathology	Result	Remarks
24. Franke[20]	1929	?	?	-	-	-	+	Peritonitis.	Perforated ulcer. Peritonitis.	Ulcer in ileac mucosa. Cap of diverticulum lined with gastric mucosa.	Died	
25. Winkelbauer[34]	1929	2	M	+	+	+	-	Intussusception.	Indurated Meckel's diverticulum.	Shallow ulcer near island of gastric mucosa.	Recovered	
26. Schmidt[54]	1930	18	M	-	-	-	+	Peritonitis.	Two chronic ulcers at neck, one perforated.	Ulcers at junction of gastric and ileac mucosa.	Recovered	
27. Aschsner and Karelitz[2]	1930	15 mo.	F	+	+	-	-	Ulcer of Meckel's diverticulum. Intussusception.	Penetrating marginal ulcer. Mesenteric adenitis.	Ulcer in ileac mucosa at edge of gastric mucosa.	Recovered	
28. Aschsner and Karelitz[2]	1930	2	M	+	+	-	-	Intussusception.	Peptic ulcer at base.	Ulcer at junction of gastric and ileac mucosa.	Recovered	
29. Fevre, Patel and Lipart[9]	1930	7	M	-	+	+	+	Perforated appendix.	Perforated ulcer at base. Peritonitis.	Chronic ulcer; duodenal ulcer present.	Recovered	
30. Fevre, Patel and Lipart[9]	1930	5 mo.	M	+	+	+	+	Intussusception.	Perforated ulcer at base. Peritonitis.	Ulcer at junction of gastric and ileac mucosa.	Recovered	
31. Haberer[28]	1930	13	M	+	+	+	+	Postoperative appendiceal abscess.	Perforated ulcer with local abscess.	Ulcer at junction of gastric and ileac mucosa.	Recovered	Operated upon one month previously for appendicitis.
32. Haberer[28]	1930	21	M	-	+	-	-	Bleeding duodenal ulcer.	Two duodenal ulcers; bleeding diverticular ulcer.	Gastric mucosa present.	Recovered	
33. Cobb[12]	1931	18	M	+	+	+	+	Acute appendicitis.	Punched-out, perforated marginal ulcer walled off.	Ulcer at margin of gastric and ileac mucosa.	Recovered	
34. Tavernier and Guilleninet[57]	1931	10	F	-	+	-	-	Possible ulcer of Meckel's diverticulum.	Small ulcer at base.	Gastric mucosa distal to ulcer.	Recovered	Preoperative x-ray series negative.
35. Lindau and Wulff[39]	1931	15	M	+	-	+	+	Peritonitis.	Peritonitis. Perforated ulcer found at autopsy.	Chronic perforating ulcer adjacent to gastric mucosa.	Died	
36. Greenwald and Steiner[24]	1931	15 wk.	M	+	+	+	+	Perforated ulcer of Meckel's diverticulum.	Punched-out perforation near base.	Areas of gastric mucosa.	Died	
37. Beuchner[8]	1931	8	F	-	-	-	-	?	Chronic ulcer at neck of diverticulum.	Gastric mucosa present.	Recovered	
38. Hudson and Koplik Case 12[32]	1932	6	M	+	+	+	+	Peritonitis.	Perforated ulcer. Peritonitis.	Ulcer at junction of gastric and ileac mucosa.	Died	
39. Hudson and Koplik Case 31[32]	1932	5½	M	-	+	+	+	?	Sigmoid full of blood. Inflamed diverticulum.	Inflammation and superficial ulceration; gastric and ileac mucosa present.	Recovered	Perforated while under observation.
40. Vaughn and Singer[53]	1932	7	M	+	+	+	-	Acute appendicitis.	Walled-off perforation.	Ulcer at junction of gastric and ileac mucosa.	Recovered	
41. Mason and Graham[45]	1932	9 mo.	M	+	-	-	+	?	Meckel's diverticulum.	Chronic ulcer at junction of gastric and ileac mucosa. Pancreatic cells.	Recovered	
42. Houdil and Marty[30]	1932	7	M	+	+	+	-	Peritonitis, appendiceal.	Adherent mass. Lentil-shaped perforation at base.	Gastric mucosa present.	Died	
43. Wulff[66]	1932	16	F	?	?	?	-	?	Chronic peptic ulcer at base.	Gastric mucosa present.	Recovered	
44. Hudson[33] Case 1	1933	11	M	+	+	+	-	?	Blood filled diverticulum.	Ulcer at junction of gastric and ileac mucosa.	Died	
45. Hudson[33] Case 3	1933	6 mo.	M	+	+	+	+	?	Perforation of diverticulum. Peritonitis.	Gastric mucosa present.	Died	
46. Dragstedt[16]	1933	17	M	-	+	+	-	Peritonitis.	Chronic perforating ulcer.	Diverticulum lined with gastric mucosa.	Recovered	
47. Johnston and Renner[26]	1933	18 mo.	M	+	+	-	+	Ulcer of small intestine. Acute appendicitis.	Acute lentil-shaped perforation at base sealed against abdominal wall.	Ulcer at junction of gastric and ileac mucosa.	Recovered	
48. Johnston and Renner[26]	1933	15 mo.	F	-	+	+	-	Ulcer of Meckel's diverticulum.	Superficial ulcer at base.	Superficial shallow ulcer at junction of gastric and ileac mucosa.	Recovered	
49. Chesterman[11]	1935	22	M	+	+	+	-	Exploratory laparotomy.	Meckel's diverticulum and tuberculous glands.	Ulcer at junction of gastric and ileac mucosa.	Recovered	
50. Chesterman[11]	1935	11	F	+	+	+	-	Peptic ulcer of Meckel's diverticulum.	Peptic ulcer of Meckel's diverticulum.	Gastric mucosa.	Recovered	
51. Chesterman[11]	1935	6	F	-	+	+	-	Possible peptic ulcer of Meckel's diverticulum.	Peptic ulcer of Meckel's diverticulum.	Gastric mucosa.	Recovered	

CHART (Continued)

REVIEW OF HISTOLOGICALLY PROVED CASES

Author	Year	Age	Sex	Pain	Intestinal Hemorrhage	Acute Abdomen	Preoperative Diagnosis	Gross Findings	Histologic Findings	Result	Remarks
52. Chesterman[11]	1935	16	M	+	+	+	Peptic ulcer of Meckel's diverticulum.	Peptic ulcer of Meckel's diverticulum.	Gastric mucosa.	Recovered	
53. Cobb[13]	1936	10	M	+	+	+	Perforated ulcer of Meckel's diverticulum. Appendicitis.	Acute appendicitis and Meckel's diverticulum.	Gastric mucosa with small peptic ulcer.	Recovered	
54. Cobb[13]	1936	1½	M	+	+	+	Ruptured viscus.	Perforated Meckel's diverticulum. Peritonitis.	Gastric mucosa with small peptic ulcer.	Died	
55. Brown and Pemberton[7]	1936	6	M	−	+(?)	−	Meckel's diverticulum.	Meckel's diverticulum.	Diverticulitis with gastric mucosa.	Recovered	Previous appendectomy.
56. Brown and Pemberton[7]	1936	7	F	+	+(?)	−	Meckel's diverticulum (?).	?	Gastric mucosa with inflammation.	Recovered	
57. Brown and Pemberton[7]	1936	9	F	+	+(?)	−	Meckel's diverticulum or polyp.	....	Gastric mucosa with inflammation.	Recovered	
58. Brown and Pemberton[7]	1936	15	M	+	+	..	Lesion of small bowel.	....	Gastric mucosa but no acid cells.	Recovered	Previous appendectomy.
59. Brown and Pemberton[7]	1936	22	M	+	+	..	Meckel's diverticulum.	....	Ulcer at base of diverticulum containing gastric mucosa.	Recovered	
60. Mallory[43]	1936	10 mo.	F	+	+	−	Ulcer of Meckel's diverticulum.	Meckel's diverticulum.	Gastric mucosa present.	Recovered	
61. Mallory[44]	1936	22	M	+	+	−	Meckel's diverticulum.	Ulcer at base of Meckel's diverticulum.	No gastric mucosa seen.	Recovered	
62. Greenblatt, Pund, and Chaney,[23]	1936	47	M	+	−	−	Duodenal ulcer with adhesions.	Diverticulum with superficial erosion.	Gastric mucosa and pancreatic tissue.	Recovered	
63. Greenblatt, Pund, and Chaney,[23]	1936	23	F	+	+	−	Diverticulitis; postoperative adhesions.	Meckel's diverticulum.	Gastric mucosa.		
64. Greenblatt, Pund, and Chaney,[23]	1936	28	F	+	+	+	Intestinal obstruction.	Intussusception of ileum; Meckel's diverticulum with ulcer.	Gastric mucosa.	Recovered	
65. Breccia[6]	1936	23	M	+	+	+	Acute appendicitis.	Meckel's diverticulum with ulcer.	Gastric mucosa and duodenal glands.	Recovered	
66. Bagwell (Curd)[4]	1936	?	M	?	?	+	Perforated duodenal ulcer.	Meckel's diverticulum.	Gastric mucosa.	Recovered	
67. Klingenhaden[35]	1936	3½	M	+	+	−	Ulcer of Meckel's diverticulum.	Ulcer at base of a Meckel's diverticulum.	Gastric mucosa.	Recovered	
68. Bunne[9]	1936	7 mo	M	−	+	−	Rectal bleeding of undetermined etiology.	Ulcer of a Meckel's diverticulum.	Gastric mucosa.	Recovered	
69. Bunne[9]	1936	3½	F	+	+	−	Ulcer of Meckel's diverticulum.	Ulcer of a Meckel's diverticulum.	Gastric mucosa.	Recovered	
70. Thompson[59]	1937	14	M	+	+	+	Acute appendicitis.	Intramesenteric diverticulum.	Ulcer of gastric mucosa.	Recovered	
71. Thompson[59]	1937	8	F	−	+	−	Ulcer of Meckel's diverticulum.	Ulcer of Meckel's diverticulum.	Gastric mucosa.	Recovered	Diagnosis of Henoch purpura two years previously.
72. Mallory[44]	1937	7	F	+	+	+	Meckel's diverticulum with ulcer.	Ulcer of Meckel's diverticulum.	Gastric mucosa.	Recovered	
73. Higgins and Gundy[29]	1937	6	M	−	+	+	Bleeding Meckel's diverticulum.	Peptic ulcer of Meckel's diverticulum.	Peptic glands.	Recovered	
74. Collins[14]	1937	22	M	−	+	+	?	Peptic ulcer in tip of Meckel's diverticulum.	Gastric mucosa.	Died	Hemophilia; poor operative risk
75. Mecray et al.[46]	1937	4	M	+	−	+	Acute appendicitis.	Perforated ulcer of Meckel's diverticulum.	Gastric mucosa.	Recovered	
76. Gabriel[21]	1937	22	M	−	+	−	Meckel's diverticulum.	Peptic ulcer of Meckel's diverticulum.	Gastric and pancreatic cells.	Recovered	
77. Matt and Timpone	1937	16	F	+	+	−	Bleeding peptic ulcer of a Meckel's diverticulum.	Peptic ulcer of Meckel's diverticulum.	Eroded small vessel; gastric mucosa.	Recovered	

N. B.: only histologically proven cases have been included in this summary.

it must only be considered as a minor component of the symptom-complex.

The treatment of peptic ulcer of a Meckel's diverticulum is surgical. The mortality rate following operation is approximately 4 per cent while the mortality following operation after the ulcer has ruptured is considerably over 50 per cent. Only the autopsy room sees those cases of peptic ulcer of Meckel's diverticulum which have been undiagnosed.

If the patient is markedly anemic, laparotomy should be deferred until one or more blood transfusions have decreased the risk of operative shock. Spectacular changes in the hemoglobin concentration can follow the administration of even a single transfusion. In our case the hemoglobin percentage rose from 46 per cent to 83 per cent nine days later.

If a reasonable doubt exists whether the condition is ulcer of the upper or lower intestinal tract, a gastrointestinal series can be done; however, only the first series of plates delineating the shape of the stomach and duodenum are necessary for the purpose of differential diagnosis.

The patient should receive an extremely restricted diet if he is to be observed or prepared for operation over a period of several days or more. The delay should be as short as possible because of the possibility of rupture of the ulcer during this period.

For reasons which we have stated previously, ileac resection is preferable to simple diverticulectomy followed by some type of purse-string suture. The type of anastomosis may depend only upon the individual preference of the surgeon. We prefer lateral anastomosis. Likewise, whether or not temporary proximal ileostomy is done remains at the discretion of the operator.

The postoperative care is obviously that of any intestinal anastomosis with the possible exception that more blood may be given by transfusion.

CONCLUSIONS

Lesions of a Meckel's diverticulum are present sufficiently often that the possi-

bility of their presence should be considered in every case presenting unusual gastrointestinal symptoms. Happily an examina-

Fig. 1. Segment of ileum with diverticulum everted to show the contrasting mucosa.

tion of the number of reported cases appearing from year to year shows that this dangerous anomaly is becoming more frequently diagnosed and treated before perforation has forced the puzzled surgeon to perform an "exploratory laparotomy."

Although a routine examination for a possible diverticulum can be quickly and easily made during any laparotomy we do not agree with Greenblatt et al. in that it should be removed, if present, unless the patient has exhibited signs or symptoms referable to the anomaly or if the diverticulum shows upon inspection evidence of being the site of one of its forms of pathology.

The presence of the hemorrhage-age-pain trilogy should point to the diagnosis of diverticular peptic ulcer as certainly as pain-vomiting-fever-leucocytosis indicates an appendicular lesion. The history of hemorrhage alone, if of the sudden, massive, recurrent type, and even if absolutely painless, should suffice to require the positive exclusion of a lesion of Meckel's diverticulum.

SUMMARY

1. Uncomplicated Meckel's diverticulum occurs in from 1.5 to 3 per cent of all persons. Approximately 25 per cent of

these contain heterotopic tissue which possesses the potentiality of becoming ulcerated.

massive bleeding from the rectum. Two days previously she had developed a dull cramp-like pain in the left lower quadrant and on the same
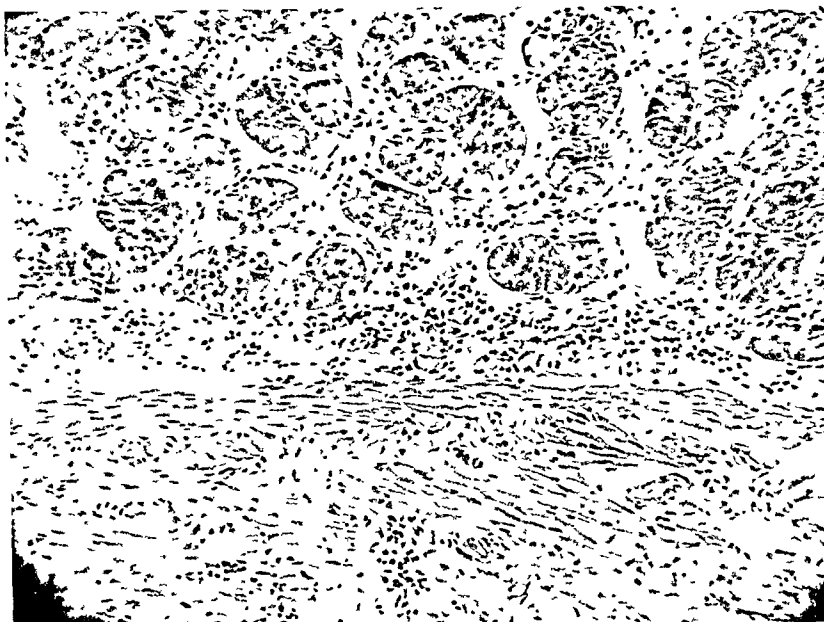
FIG. 2. Section through diverticulum showing polygonal cells similar to the parietal cells of gastric stroma. ($\times$ 200.)

2. Theories explaining the presence of heterotopic tissue are stated. The dysembryoma theory apparently satisfactorily explains the conditions found in lesions of Meckel's diverticulum.

3. The pathologic classification of lesions of Meckel's diverticulum as proposed by Greenblatt and his associates is charted. The possibility of clinical diagnosis of lesions of the umbilical and peptic groups is illustrated.

4. The diverticular ulcer trilogy of hemorrhage-age-pain is stressed and evidence supporting their reliability is presented.

5. A review of the literature to 1938, to which the authors add a case diagnosed correctly preoperatively, is presented.

6. Diverticulo-ileac resection following adequate preoperative preparation of the patient, offers an almost perfect surgical record.

CASE REPORT

I. D., a 16 year old Italian girl, was admitted to the medical service of Dr. Joseph W. Diamond March 25, 1937, complaining of sudden

evening she passed a large quantity of dark red blood and clots which contained no mucus. On the day before admission to the hospital she had seven bowel movements all of which were composed of pure blood. She had no tenesmus or griping and the abdominal pain had diminished in severity since the time of onset. She had no hemorrhage elsewhere and no petechiae had been noticed. She stated that she had had similar attacks four times during the past three years, the last having occurred eleven months before; since then she had been in good health and her stools had been normal. She had a good appetite, could eat any type of food and never had any symptoms referable to the gastrointestinal tract. She had had the usual diseases of childhood, but otherwise her past history was negative. She began to menstruate at the age of 12, her menses appearing regularly every thirty days and lasting five days. Her last menstrual period began March 5, 1937 and was not unusual.

The patient was well developed, well nourished, but markedly anemic. She had no elevation of temperature, and her pulse and respirations were normal.

The sclerae were extremely pale, as were the lips and buccal mucosa; the nasal mucosa was

slightly inflamed and covered with a scanty mucopurulent exudate. No hemorrhagic manifestations were found in the nasopharyngeal or buccal mucosa.

The chest and heart were normal. The blood pressure was 72/42, the pulse 78 and respirations were 18 per minute.

The abdomen was flat and unscarred. The liver was not palpable, but the spleen descended two fingers below the costal margin during deep inspiration (?) and was slightly tender. No other masses were palpable, but deep pressure over the left lower quadrant caused slight tenderness. Rectal examinations, digital and visual, were negative save for the presence of blood on the mucosa of the rectum and sigmoid colon. The extremities were normal. Neurologic examination yielded normal responses.

Radiographic examination of the gastro-intestinal tract following a barium meal showed no evidence of any organic pathology; examination following a barium enema was also negative.

The red blood cell count was 2,740,000 per cu. mm. with a hemoglobin concentration of 46 per cent and a color index of 0.85 per cent. There was marked anisocytosis, moderate piokilocytosis, and slight polychromatophilia. There were 210,000 platelets per cu. mm. of blood. The leucocyte count was 11,700, of which 80 per cent were polymorphonuclear neutrophiles, 1 per cent eosinophiles and 19 per cent small lymphocytes. Bleeding time and clotting time were normal and clot retraction was normal, yielding a firm clot. The capillary resistance test was negative. Blood Wassermann was negative. There was 10.3 mg. of calcium and 3.8 mg. of phosphorus per 100 c.c. of blood. Urinalysis was essentially negative and examination of a stool specimen composed almost entirely of blood was otherwise negative.

The possibility of ulcer of a Meckel's diverticulum was suggested by Dr. M. B. Genaur and diverticulectomy was decided upon. A transfusion of 500 c.c. of whole blood was given by the Scannell method and the patient was placed on massive doses of iron and liver and given a light low residue diet.

On April 10 the erythrocyte count was 4,570,000 with a hemoglobin concentration of 83 per cent. The patient was transferred to the surgical service and three days later laparotomy was performed. The abdomen was opened through a right paramedian incision and a diverticulum was encountered on the anti-mesenteric surface of the ileum approximately 14 inches proximal to the ileocecal junction. A segment of ileum 4 cm. long and containing the diverticulum was removed and lateral anastomosis was done. A temporary ileostomy was made proximal to the line of anastomosis and the abdomen closed following removal of an apparently chronically inflamed appendix.

The postoperative course of the patient was uneventful. She expelled the intraluminal portion of the ileostomy tube on the eleventh postoperative day and was discharged cured fifteen days following operation. At the present time she is in excellent health and is enjoying a normal life in every respect.

The pathologic specimens represented part of the ileum, measuring 9.5 cm. in length. At the mid-portion, a large cherry-sized bulging diverticulum was present. The serosa was shiny throughout. The mesenteric fat extended to the diverticulum and showed a few petechial hemorrhages, while the mucosa of the ileum was covered with bloody mucus. A probe passed easily into the diverticulum whose opening was 20 mm. in diameter. At the base a punched out defect 4 mm. wide was covered by blood clot. The diverticulum itself was filled with mucus and its mucosa was markedly polypous.

Section through the diverticulum showed the mucosa lined by mucus cells with abundant mucus formation. These cells formed ramifying gland-like structures dipping deeply into the stroma. In the deeper layer of the stroma the glands were lined by large polygonal cells with abundant eosinophilic protoplasm with the typical appearance of parietal cells of the stomach. In the basal portions of the glands there were also cells of granular protoplasm similar to septic cells of the stomach. Section through the small intestine showed the typical villi and mucosa of the ileum.

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Wounds of the abdomen which are inflicted by the modern missiles of warfare exert their lethal effect by producing hemorrhage or by engendering sepsis, either through infection carried in with the fragment of high explosive or through damage to the hollow viscera of the belly, especially of the gastro-intestinal tract. The bowel is that portion of the alimentary tube most liable to be wounded, and is damaged nine or ten times as often as is the stomach.

From—"War Wounds and Air Raid Casualties" (Lewis).

PTOSIS OF CECUM AND ASCENDING COLON

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DEFINITION

PTOSIS may be defined as a prolapse downward of one or more of the abdominal viscera to such an extent as to change materially the normal relationship to one another and to the adjacent structures, especially when the subject is in the erect posture.

ETIOLOGY

1. Predisposing causes:
   - (a) Arrested development
   - (b) Maldevelopment
   - (c) Skeletal posture, shape of abdominal cavity, strength of abdominal wall
2. Exciting causes:
   - (a) Intra-abdominal pressure
   - (b) Occupation

To understand maldevelopments it is essential to have at least a rudimentary knowledge of normal development. The alimentary tract is developed from the entodermic vesicle of the zygote.[2] This vesicle becomes divided into an intra-embryonic and an extra-embryonic portion. The intra-embryonic portion becomes the alimentary tract and appendages. About the fifth week the alimentary tract can be differentiated into three main subdivisions, the foregut, the midgut and the hindgut. The divisions are made on form, blood supply, and function. Each part has a separate blood supply. The foregut forms the gastrointestinal tract below the ampulla of Vater to the distal portion of the transverse colon with its circulation supplied by the superior mesenteric artery. The hindgut is caudal to the transverse colon and is supplied by the inferior mesenteric artery. The function of the foregut is digestive; of the midgut absorptive; and of the hindgut excretory. Developmental error is common in the midgut while in the foregut and hindgut malformation is of rare occurrence.

The vitelline duct ordinarily becomes detached about the fourth week of life. The rapid increase of the size of the liver of the midgut is out of proportion with that of the rest of the abdomen. The peritoneal cavity cannot hold all the structures, and the midgut is therefore pushed out through the umbilicus, producing a physiologic umbilical hernia. The apex of the herniated loop is at the former attachment of the vitello-intestinal duct and the termination of the superior mesenteric artery, which originally was the right omphalomesenteric artery. The artery lies in the mesentery and sends branches to the anterior and posterior segments. The mesentery of the herniated loop Dott divides into a pre-arterial and a post-arterial portion. The gut in this stage occupies a sagittal plane. The rotation of the midgut occurs between the fifth and tenth week and may be divided into three stages. In the first stage the pre-arterial segment is on the right side and the post-arterial on the left. The rotation is necessitated by the enlargement and the descent of the liver and pressure of the umbilical artery. The pre-arterial portion and the mesentery become disproportionately long. An enlargement in the post-arterial segment can be seen in an embryo from twenty-seven to thirty days old. This is the beginning of the development of the cecum and appendix.

The second stage of rotation begins about the beginning of the tenth week when the gut returns to the abdomen. Mall suggests that the increase in the length of the loops and their rotation in the abdominal cavity produce enough traction to replace the extra-abdominal intestinal loops within the abdomen. The

internal orifice of the umbilical canal is comparatively small, making it impossible for the entire contents of the hernia to return to the abdominal cavity en masse. The greater size of the cecum may offer sufficient resistance to retain it in the hernial sac, thus permitting the pre-arterial segment to return first. In doing so, it passes behind the superior mesenteric artery which extends from the aorta to the umbilicus. The small intestine as it enters the abdomen pushes the intra-abdominal small gut and mesentery, which has been occupying the midline, to the left and posterior portion of the abdominal cavity. The cecum is the last to be reduced into the abdominal cavity. It occupies a position in the region of the umbilicus, anterior to the small intestines and superior mesenteric artery and beneath the liver. The cecum continues to descend to lie in the normal adult position in the ileocecal fossa. In the twelfth week the superior mesenteric artery, which is brought to the iliac region by the migration of the cecum, lies in intimate contact with the posterior parietal peritoneum and becomes adherent from above downwards to the posterior abdominal wall. Along the right side of the artery the ascending colon and cecum become fixed. The post-arterial mesenteric segment persists as the mesocolon. The pre-arterial segment remains free as the mesentery of the small intestine. The mesentery of the hindgut becomes obliterated by fusion along the midline with the posterior parietal peritoneum.

DEVELOPMENTAL ANOMALIES

Malrotation and non-rotation depend upon the sequence with which the intestinal loops return to the abdomen. As a result of non-rotation the small intestine lies chiefly to the right. The duodenum passes to the right of the superior mesentery artery. The colon is located entirely on the left side and does not cross the superior mesenteric artery. Normal fixation or absence of fixation, is frequently encountered, producing an abnormally

mobile sigmoid or cecum. Non-rotation is the result of a return of the cecum and the post-arterial segment to the abdominal cavity before return of the pre-arterial segment.

Reverse rotation may take place but it is of little significance clinically, because the various parts of the intestinal tract have normal relationships, except that they are in reverse. In malrotation of the midgut, the pre-arterial segment of the midgut passes in front of the superior mesenteric artery and the posterior segment including the cecum also passes in front of the origin of the artery. The mesentery does not become adherent to the posterior abdominal wall. The duodenum and ileum lie side by side in front of the superior mesenteric vessels.

The various congenital anomalies are due to the variation in the size of the embryonic umbilical orifice. Most clinical anomalies, however, arise from faulty third stage rotation. The third stage rotation consists largely of fusion of the various mesenteries with the posterior parietal peritoneum, making the various parts of the gastrointestinal tract less mobile. Early fixation or deficient fixation with the exception of the elongation of the colon produce definite anomalous conditions. The most important of these, pathologically, are those resulting from deficient fixation of the colon.

The position of the retrocecal appendix can easily be explained if one remembers the descent and fusion of the cecum and ascending colon to the parietal peritoneum. The appendix can be turned back behind the descending cecum, becoming enclosed in the mesocolon with the parietal peritoneum. On the other hand, if the appendix takes a position anterior to the cecum in the descent of the colon it will have a free position in the abdominal cavity.

EXCITING CAUSES

If the occupations of ptotic people require that they be on their feet and

especially if they have to do lifting, the condition progresses from bad to worse. Through the loss of weight and of the fat

supporting the abdominal viscera is almost a negligible one.

Long continued intrathoracic conditions,
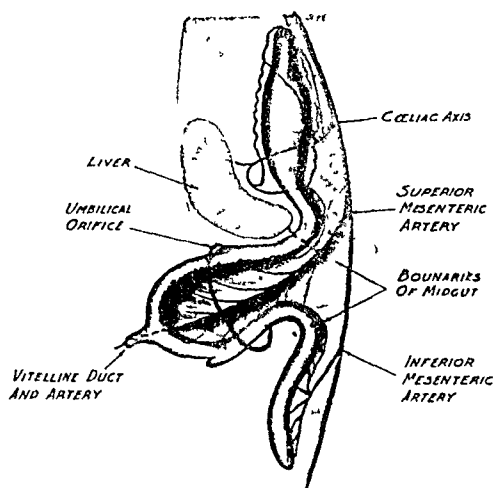
FIG. 1.
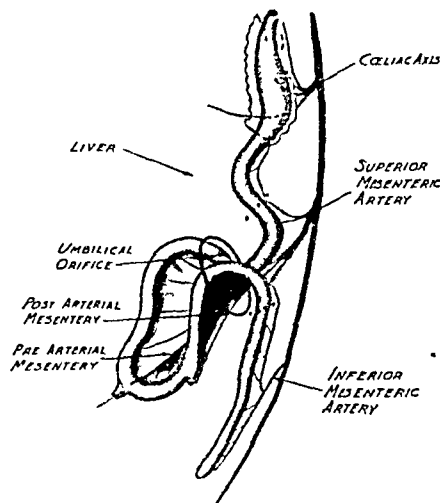
FIG. 2.
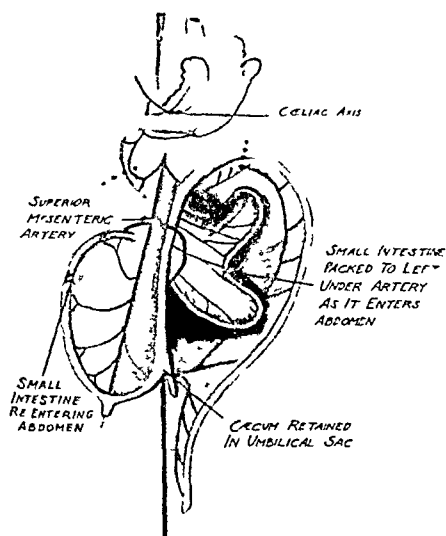
FIG. 3.

FIG. 4.

FIGS. 1 TO 4. Stages of development and rotation of the midgut.
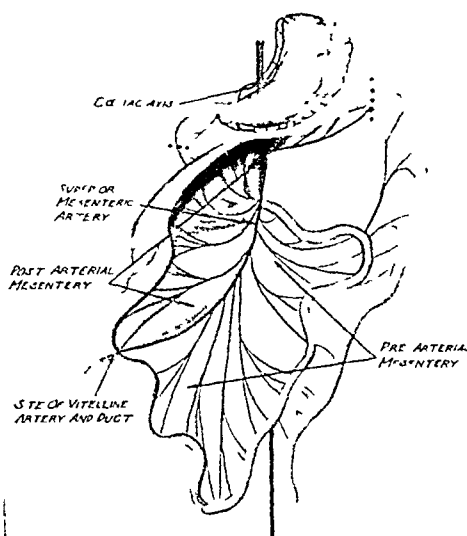
in the meshes of the mesentery greater elongation occurs, causing a sagging of the abdominal viscera.

A relaxed abdominal wall after long wasting disease may be followed by a visceroptosis. The relaxation of the lateral muscle groups, that is, the transversalis and the oblique, than that of the recti abdominis, which determines the condition. The part played by the pelvic floor in

as large exudates, new growths and, at times, pneumothorax may cause ptosis of the abdominal viscera. In this condition the diaphragm is fixed in the inspiratory position and thus renders its muscular opponents, the muscles of the abdominal wall, useless. Dr. Arthur Keith has made an embryologic and anatomic study of this condition and states that the abdominal muscles in thoracic respiration act as

opponents to the diaphragm. In a shelf-like manner they support the abdominal viscera during inspiration and aid the diaphragm in the elevation of the ribs and thorax. In relaxation of the lateral muscles of the abdominal wall this normal relationship is lost.

The tendency to ptosis often runs in families, various members showing a phthisical chest and giving a history of ancestral tuberculosis.

PATHOLOGIC ANATOMY AND PHYSIOLOGY

In the normal position of the cecum and ascending colon in the ileocecal fossa, where proper fusion of the post-arterial segment has taken place, they get support from what Coffey calls the iliopsoas shelf. The center of gravity passes through the ascending colon and cecum, well posterior to the anterior border of the psoas muscle. In deficient fusion with a long mesocolon the weight of the intestine forces it into the pelvis, causing a gradual elongation of the mesentery. Nature attempts to correct the defect by laying down a membrane which gradually becomes more dense, and often forming bands which become painful when pulled upon by the distended cecum. These bands produce stagnation by kinking the ascending colon in the region of the hepatic flexure.

A child born with a defective prenatal fixation of the abdominal viscera will have gastrointestinal upsets from birth on. After adolescence, with the greater activity of the child, a gradual development of the typical ptotic figure is the rule. The colon, dropping from its normal location, slides off the psoas shelf into the pelvis. The kidney fossa becomes shallower. The kidney often slides down. The drag on the bottom of the stomach causes displacement of the pyloric end of the stomach, retarding the gastric contents. This causes indigestion with subsequent loss of flesh and further reduces the visceral support by decreasing the intra-abdominal pressure. Gas and fluid in the gastrointestinal tract are increased. The stretching of the gastro-intestinal muscles causes a gradual reduction of peristalsis. The efficiency of digestion is generally lowered. The abdominal organs tend to gravitate more and more to the lower portion of the abdomen and pelvis.

The drag on the diaphragm and the fascia attached to the ribs causes the lower portion of the chest to become narrow and the angle of the ribs with the vertebrae to become sharper. The lower end of the spine and the pelvis are tilted backwards in an effort to hold the load by means of the thoracic fascia.

The normal curvature of the spine disappears, and the spine becomes relatively straight with the lower end tilted relatively forward and upward and the upper part backward.

The drag on the visceral nerves and sympathetic plexus materially affects the nervous stability of the patient, and these individuals are often classified as neurasthenics. The intestinal stasis and resulting autointoxication extending over years causes a depletion of the adrenals with a gradual lowering of the blood pressure, increasing weakness and making for easy exhaustion on physical and mental exertion.

SYMPTOMATOLOGY

The discussion of symptoms is here limited to those of right sided abdominal ptosis only, the ptosis of the cecum and ascending colon. This condition is, practically without exception, due to a congenital defect, the cecum and ascending colong having failed to fuse properly with the posterior parietal peritoneum. The symptoms generally appear at the time of adolescence and are often diagnosed as intestinal colic. There are attacks of right sided abdominal pain which may last only a short time or may last for days, with rest in bed necessary before they subside. As time goes on the cecum and ascending colon become larger and their contents are less easily expelled.

Nature meanwhile tries to come to the rescue by laying down supporting mem-

branes along the line of stress from the right lateral wall of the abdominal cavity to the cecum and ascending colon. These

FIG. 5. Tabulation of cases with prominent symptoms, treatment and result.

membranes stand out as dense white bands, often kinking the bowel in the region of the hepatic flexure, still further impeding the movements of the intestinal contents. The appendix in these cases is apt to become infected because of the stagnation in the colon. Removal of the appendix gives little or no relief.

There is vomiting after the pain has started, not at the beginning as in an attack of acute appendicitis. There may be fever of a degree or so if the stasis is considerable.

On physical examination we often find a palpable cecum and an ascending colon which contains gas and fluid. There is tenderness along the attachment of the membranous bands. In severe cases we find a ptotic figure and signs of neurasthenia.

A barium meal shows a low lying cecum and often a retention of barium for days. A barium enema will show the same and, if there are tight bands, delay of the barium enema at the region of the hepatic flexure can be seen.

DIAGNOSIS

In the differential diagnosis we may consider all right sided abdominal conditions which give rise to pain, vomiting and toxemia, as well as right sided kidney lesions and, in women, right pelvic lesions. The succussion splash in the cecum is said to be pathognomonic of a redundant cecum with stasis. Gastrointestinal x-ray studies are conclusive, but a diagnosis can generally be made without the aid of the x-ray.

The history of right sided abdominal pain from the time of adolescence which is apt to get worse on physical exertion especially when the patient has to be on his feet for any length of time is characteristic. This pain may become, worse with each attack. Pain occurring during or after an evening of dancing is very suggestive. The location of the pain is higher than the average appendix. It is described as a dragging or a heavy pain in the right side or, if partial obstruction has occurred, as colicky.

PROGNOSIS

Due to the stasis and irritation produced by decomposition a colitis is set up which in advanced cases spreads from the ptotic portion of the colon. The symptoms are thus far in excess of what one would expect from the original pathology. After the correction of the pathologic anatomy the colitis must be treated. Otherwise, the prognosis is good.

TREATMENT

Medical management consists in relieving the intestinal stasis and attempting to shorten the mesentery by deposition of fat in the mesenteric meshes. The patient should be kept in bed with the foot of the bed raised and put on forced feeding, sometimes for months. A gain in weight may relieve all symptoms. The shortened mesentery supports the intestines well enough to prevent kinking. If the weight can be maintained after a return to usual activities, good health may continue, but generally a gradual loss of weight causes return of the symptom complex.

Belts and abdominal supports may be valuable in gastroptosis and general ptosis

but I believe they are worse than useless in right sided ptosis, except for their psychic effect. Surgical correction should be carried out before a marked colitis has set in.

Surgical treatment is in most cases successful. Preoperative management requires emptying of the colon with a mild laxative and enemata. Hospitalization for some days prior to the operation is advisable. Intraperitoneal vaccination is also valuable, especially if there is concomitant colitis. In debilitated patients, a build up program before operation should be carried out. Transfusion is helpful.

Spinal anesthesia gives excellent relaxation and should allow sufficient time. The operation can be performed in less than an hour. Ether anesthesia, where there is no contraindication, is satisfactory.

A right pararectus incision is made, of sufficient length to enable one to expose the hepatic flexure and the cecum. The bands and adhesions are released and the appendix is removed. An incision is made lateral to the cecum and ascending colon as high as the hepatic flexure, this being the lateral leaf of the ascending mesocolon. The peritoneum is reflected laterally, exposing the perirenal fat, practically a bloodless dissection. Care is taken not to injure the ureter.

Sutures of chromic catgut or dulax are passed through the edge of the reflected peritoneal fold, the first through the lower end of the reflected fold and the region of the appendiceal stump. The following sutures are inserted $\frac{1}{2}$ inch to 1 inch apart through the anterior longitudinal band of the cecum and ascending colon. I have found it advantageous to leave the sutures untied until they are all inserted. When the sutures are tied the pendulous cecum and ascending colon roll into the denuded space where they soon will become firmly attached. As there generally is a coexisting ptosis of the right kidney, the kidney will become fixed in its proper position. At times the bands and membranes which have been formed in nature's attempt to relieve the pathologic condition can be

used to strengthen the anchoring of the ascending colon. If there still remains a sharp kinking at the hepatic flexure the incision can be curved around and the condition corrected by suitably placed sutures.

In cases where there is a long transverse colon which falls down into the pelvis, dragging on the hepatic flexure, I anchor it to the anterior abdominal wall medial to the hepatic flexure. Sutures are inserted through the root of the omentum and parietal peritoneum of the anterior abdominal wall. This is what Coffey describes as his hammock operation. It can be extended right across the abdominal cavity if necessary and will give support to a ptotic stomach.

These patients have remarkable little postoperative disturbance. The relief of the partial obstruction and the absence of intestinal toxemia give them a feeling of well being. They can leave the hospital when the wounds are healed and are permitted to resume active life as after any other similar abdominal operation. Colitis, if present, is treated by diet and other measures. The patient often expects to be completely cured and does not coöperate for a long enough time to have the colon return to normal. In cases where a portion of the colon is much involved it might be wise to do a partial colectomy.

CASE REPORTS

I have operated on twelve patients and have seen a number of other cases in which no operation has been done, but in which the diagnosis has been established by radiographic study. The twelve operations have produced highly satisfactory results, with ten complete clinical cures and two much improved. The colitis which existed in the latter two cases necessitates a more or less strict dietary regime. They are, however, relieved of the toxemia and the symptoms associated with the partial obstruction. Typical case histories are appended.

CASE I.   A. S., male, age 20, complained of right sided abdominal pain occurring at intervals of a few weeks, generally following dietary indiscretion or excessive physical exertion. The symptoms first appeared at the age of 14 and had since gradually increased in severity. Vomiting occurred with the attacks, but rarely fever. The pain lasted for two or three days. A diagnosis of appendicitis had been made.

There was a palpable cecum containing gas and fluid. The leucocyte count was 10,000, the red count 4,400,000. A barium meal revealed a low lying cecum with stasis and probable kinking at the hepatic flexure.

At operation the appendix was found to be normal, but there was a well developed Jackson's membrane with tight bands at the hepatic flexure. The cecum and ascending colon were distended and pendulous. The appendix was removed and a Waugh operation was performed. The postoperative course was uneventful and complete recovery followed.

CASE II.   A. N., male, age 28, had pain in the abdomen especially in the right side, fever, vomiting, constipation, attacks of diarrhea, and a rash which he had been told might be due to syphilis.

There was a palpable mass in the right side of the abdomen, movable from side to side, and containing fluid and gas. The temperature was 103°, the Wassermann negative, leucocytes 12,000. The urine was normal. A diagnosis of intestinal stasis with toxemia was made. The intestinal stasis was relieved, the symptoms disappeared, and the patient returned to work.

One month later the condition recurred, and the patient was operated on. A large and pendulous cecum and ascending colon were found with bands between the ascending colon and the lateral wall of the abdomen. The appendix showed no gross changes. The appendix was removed and a Waugh operation performed. Smooth recovery ensued, with no return of symptoms.

CASE III.   N. A., female, age 25, had had periodic right sided abdominal pain for the previous ten years. It was apt to come on after exertion, such as dancing, but disappeared overnight or in a day or two. She was easily fatigued, had frequent headaches and was physically below par.

Gas and fluid could be made out in the cecum and ascending colon. There was tenderness along the ascending colon. A barium meal revealed a pendulous cecum with delayed emptying of the cecum and ascending colon. Tight bands at the hepatic flexure were found at operation. The appendix was apparently normal. It was removed, the bands cut, and a Waugh operation performed. Convalescence was uneventful and recovery complete.

CASE IV.   H. P., female, age 25, had abdominal pains from 12 years of age. An appendectomy was done at the age of 20, when she was told that the appendix was normal but there was some pelvic condition causing her trouble ("fibroid uterus"). Two years later she was operated on for an ovarian cyst. The right sided abdominal pain with constipation and toxemia persisted, however, and sometimes confined her to bed for days at a time. General debility and neurasthenia were noted. Gastrointestinal radiographic study revealed stasis in the cecum and ascending colon. A long pendulous cecum rested on the pelvic floor. There were probably adhesions from the previous operation. The transverse colon was long and occupied the pelvis. The colon was kinked at the hepatic flexure.

A long pararectus incision was made on the right side. There were adhesions between the cecum and the old appendectomy scar, as well as bands and membranes between the cecum and ascending colon and the right side of the abdominal wall. The colon was inflamed and edematous. The adhesions were freed and colopexy performed with the incision curving around the hepatic flexure. Sutures were placed in the root of the large omentum and to the anterior abdominal wall medial to the hepatic flexure halfway across the abdomen.

In this case there was marked colitis extending practically half way across the transverse colon and very marked at the hepatic flexure. The patient's postoperative course was smooth, a diet had to be followed. However, the stasis was relieved and if there is no dietary indiscretion the patient's health is good.

CASE V.   W. G., male, age 34, had periodic abdominal pain for several years which at times forced him to bed. He felt better when he kept his bowels moving with laxatives and enemata. He was brought to the hospital as an emergency in the middle of the night with a fever of 103, marked tenderness and rigidity over the right side of the abdomen. Tenderness was equally marked under the right costal

margins and over the right lower quadrant. Leucocytes numbered 18,000, with 90 per cent polymorphonuclears.

A long right pararectus incision made. An acutely inflamed appendix with a distended cecum and ascending colon and tight bands at the hepatic flexure causing almost complete obstruction were observed. The ascending colon was inflamed. The appendix was removed, the bands cut and a modified right sided colopexy done. A stormy convalescence for the first few days was followed by complete recovery.

CASE VI. M. C., female, age 20, had had pain since the age of 13 or 14 which had gradually grown worse. She was afraid to go to dances because of the pain which followed. The attacks were always associated with vomiting, rarely by fever. There had been considerable loss of weight. One year before an appendectomy had been performed, but a long convalescence had been followed by no improvement.

There was a palpable cecum and ascending colon, tenderness over the right side of the ascending colon. Blood and urinary findings were normal. Radiographic study showed six hour retention in the stomach, a large pendulous cecum and colon, and apparently bands at the hepatic flexure.

Operation confirmed the findings of the radiographic study. Adhesions between the pylorus, duodenum and hepatic flexure and Jackson's membrane were noted. The adhesions were released and a colopexy performed. Smooth convalescence followed.

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MESENTERIC VASCULAR DISEASE*

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THE increasing incidence of vascular disease[1] makes a better knowledge of the abdominal manifestations desirable. Recently a number of significant contributions to this subject have been made.[2,7]

In the present paper the etiology and pathology of mesenteric vascular disease are reviewed briefly, the clinical manifestations are discussed in detail and certain points in the surgical management of these cases are outlined.

The etiologic factors involved in vascular disease of the mesentery and the relative frequency of these factors as observed in the Peter Bent Brigham Hospital are shown in Tables I and II. The important fact to be noted is that arterial thrombosis accounted for over 50 per cent of the cases. This means that, except for arteriosclerosis, an obvious clinical cause for the occlusion such as a source for an embolus, cirrhosis of the liver or intra-abdominal sepsis was lacking. Moreover, in those cases in which the patient's condition was most favorable for surgical therapy, generalized arteriosclerosis was not a prominent finding. Consequently, an obvious etiologic factor should not be considered essential in the clinical diagnosis of mesenteric vascular disease.

The mechanism and pathologic anatomy of mesenteric vascular occlusion merits brief consideration. Klein's[8] careful study of the available experimental and pathologic material has shown that the closure of a mesenteric artery may be followed by three possible results, depending largely upon the site and the rapidity of onset of the occlusion and on the status of the collateral circulation. These possibilities are: (1) complete establishment of a collateral circulation which may (a) persist effectively throughout the life of the patient, or (b) subsequently break down, usually due to a progression of the occlusive process or cardiac failure; (2) intestinal symptoms, such as pain, vomiting, or obstipation due to a blood supply which is sufficient for life of the parts, but not for function; or (3) intestinal infarction with the injury varying from a moderate degree of necrosis to gangrene of the entire intestinal wall.

If a sudden occlusion occurs at the base of the superior mesenteric artery, an extensive anemic infarction of the bowel results. However, if the occlusion is sufficiently gradual in onset a completely competent collateral circulation may develop. If the occlusion occurs in a branch of the superior mesenteric artery, anywhere above the last arcade, there may be no symptoms if the occlusion is gradual in onset, but if, as usually happens, the occlusion occurs suddenly there is an associated vascular spasm of the collateral circulation and an anemic infarction of a portion of the bowel results. However, in this location although the initial infarction is anemic in character it subsequently becomes blood soaked and hemorrhagic, because as the vascular spasm subsides, there is a back flow of blood from the collateral circulation into the involved area. As the majority of arterial occlusions are in this location, one usually finds at operation or necropsy an edematous, swollen, hemorrhagic bowel. Occlusions below the last arcade in most cases probably cause no symptoms. In certain instances transient symptoms, falling into the group of "so-called abdominal angina" may be observed.[4] In venous occlusion there is a continued flow of blood from the arteries into the infarcted area, so that the

lesion is always of a blood soaked, hemorrhagic nature. Thus, although the mechanism is different, the final gross pathologic picture in both venous and arterial occlusion is quite similar.

TABLE I

Arterial
- (a) Embolic......
  1. Rheumatic heart disease with auricular fibrillation.
  2. Arteriosclerotic heart disease with mural thrombi in the heart or aorta.
  3. Acute endocarditis.
  4. Subacute endocarditis.
- (b) Thrombotic...
  1. Arteriosclerosis.
  2. Thrombo-angiitis obliterans.
  3. Syphilis.

Venous.
- (a) Intra-abdominal sepsis.
- (b) Vascular stasis
  1. Cirrhosis of liver.
  2. Other mechanical causes such as carcinoma, enlarged glands, etc.
- (c) Blood dyscrasias
- (d) Idiopathic (probably often a mechanical cause, such as a volvulus or internal hernia which has become reduced before operation or autopsy).

Although an occlusion of the mesenteric veins may be gradual in onset, the clinical

TABLE II

Cause	Type of Occlusion	No. of Cases
Arteriosclerosis.........	Arterial thrombotic	15
Cirrhosis of liver. . ..	Venous thrombotic	3
Idiopathic..............	Combined	3
Endocarditis............	Arterial embolic	3
Intra-abdominal sepsis....	Venous thrombotic	2
Rheumatic heart disease..	Arterial embolic	2
Thrombo-angiitis obliterans..............	Arterial thrombotic	1
Syphilis.................	Arterial thrombotic	1

picture, as will be discussed later, is sometimes as sudden in onset as in an arterial occlusion. There are two reasons for this. First, when the occlusion is extensive, there is a tremendous loss of blood from the arterial side into the involved segment so that profound shock may develop with even more dramatic suddenness than is

observed in arterial occlusion. Secondly, in low grade, slowly progressive thrombosis of the mesenteric veins, the compensatory powers of the collateral circulation may be sufficiently great to prevent symptoms up to a certain point and then collapse suddenly.[5] It can also be seen from the above discussion that arterial occlusion may be so small as to produce either very slight or very slowly progressive signs so that, as will be pointed out no fundamental distinguishing features can be expected in the clinical aspects of arterial or venous occlusion.

The anatomic distribution of the lesions in the present series is shown in Table III. The ileum alone was involved eight times; the jejunum alone, three times; and both ileum and jejunum, thirteen times. In four cases the lesion involved the colon in addition to the small bowel, and in only two

TABLE III
ANATOMIC DISTRIBUTION

	Arterial		Venous			Total
	Embolic	Thrombotic	Infectious	Vascular Stasis	Idiopathic	
Jejunum only...	0	3	0	0	0	3
Ileum only......	1	4	0	2	1	8
Jejunum and ileum..........	1	7	2	1	2	13
Jejunum, ileum and ascending colon........	2	2	0	0	0	4
Ascending colon only..........	0	1	0	0	0	1
Sigmoid colon only.........	1	0	0	0	0	1

cases was the large bowel alone involved. This distribution is quite typical and is similar to that of previously reported series. The significance of the anatomic distribution lies in the fact that the ileum is involved in over 80 per cent of the cases. This has an important bearing on the surgical approach to the lesion.

CLINICAL MANIFESTATIONS

A correct diagnosis of mesenteric vascular disease is rarely made, partly because of the comparative rarity of the condition

and partly because of certain inherent difficulties in diagnosis. A source of embolus, copious melena, a rapid fall of temperature and severe abdominal pain were at one time thought essential for a diagnosis, and even today experienced clinicians will dismiss the possibility of a vascular accident in the abdomen because "the patient is not sick enough." It cannot be emphasized too strongly that mesenteric vascular occlusion, particularly when it involves the arteries, is not always fatal and the severity and intensity of the symptoms are as variable as those of peripheral or coronary artery disease.

For convenience of discussion, the clinical manifestations of mesenteric vascular disease may be divided into three groups: Those which result from spasm or small non-fatal occlusions of the mesenteric arteries, so-called "abdominal angina" or "abdominal intermittent claudication"; those which follow an extensive occlusion of either a mesenteric artery or vein, "mesenteric vascular occlusion"; and those which are a consequence of the rupture of a sclerotic artery with intraperitoneal hemorrhage, "intra-abdominal apoplexy."

"Abdominal angina" has been described for years, but until recently, a lack of correlation between clinical inference and pathologic confirmation has prevented its acceptance as a clinical fact. Such distinguished clinicians as Sir William Osler and Sir Clifford Allbutt doubted its occurrence. However, sufficient evidence has now been accumulated to prove that vascular disease of the mesentery can cause abdominal pain in the absence of gangrene of the bowel or peritoneal irritation.[4,7,10] The exact mechanism involved in the production of abdominal angina is not definitely established. That it is due to vascular spasm seems less likely than that it is the result of a relative anoxemia from small, non-fatal occlusions. The partially occluded vessels, although sufficient for viability, are unable to meet the increased demands for blood necessary for function, exactly as in the arteriosclerotic leg which is over-exercised.[4]

The clinical importance of abdominal angina lies in the fact that it may be the precursor of extensive mesenteric vascular occlusion, and a proper appreciation of its characteristics is of importance in the early recognition of this highly fatal surgical emergency. In the cases of this type which we have had an opportunity to observe, the pain has been rather deep seated in the mid-abdomen or epigastrium, has not radiated, and has not been well localized. There has been a definite relation between the ingestion of food and the onset of pain and in three instances the patients have abstained voluntarily from heavy eating, with resultant relief. Although severe, the pain has not been associated with spasm or exquisite tenderness of the abdominal wall. Naturally, in such cases, a thorough examination of the biliary and gastrointestinal tracts by means of one x-ray is necessary. Then if the findings are negative a diagnosis of abdominal pain of vascular origin is justified.

In a previous communication[4] we have commented on the characteristics of abdominal vascular pain, pointing out that it appears to be a true visceral pain and consequently is not associated with muscular spasm or exquisite tenderness of the abdominal wall. This is of considerable importance in the early diagnosis of extensive mesenteric vascular occlusion, because the pain which occurs in the early stages of this condition differs from simple vascular pain only in its constancy. It also has the characteristics of true visceral pain as it is not associated with muscular spasm or exquisite tenderness until gangrene and peritonitis have been established. This contrast between the severity of the pain and the paucity of the physical findings is one of the most significant diagnostic features in the early stages of mesenteric vascular occlusion.

The extensive occlusion of either a mesenteric artery or vein presents a fairly definite clinical picture. Loop[9] was the first investigator to emphasize distinguishing features. He pointed out that the paramount symptoms are a disturbed function

of the gastrointestinal tract, manifested by severe abdominal pain, vomiting, difficulty in moving the bowels without complete obstruction, and shock of variable degree. He noted the absence of fever, muscular spasm, and distension. Dunphy and Zollinger[2] arrived at similar conclusions from an analysis of four personally observed cases, and made a correct preoperative diagnosis in a fifth case. This led to a successful resection. Since that time at the Peter Bent Brigham Hospital a correct preoperative diagnosis has been made in two more cases and in a third case a correct diagnosis was proved by post-mortem examination, the patient having been moribund from associated diseases.

The features which we have noted are: (1) Regardless of the duration of the attack, the clinical picture has not been typical of the common surgical emergencies. The localized tenderness of appendicitis, the rigidity of perforated ulcer and the visible peristalsis and early distention of mechanical intestinal obstruction are not present. Usually one is able to makes no definite diagnosis. However, the patients always appear ill from an abdominal lesion which simulates, but is not typical of, obstruction. (2) The character of the pain is quite out of proportion to the clinical findings and persists after ordinary measures have been instituted for its relief. (3) The only constant physical finding is deep abdominal tenderness more or less generalized with rebound tenderness referred to the point of pressure. In the later stages the signs of generalized peritonitis develop, but these should not be considered essential in the early diagnosis. (4) The leucocyte count and pulse rate are almost invariably elevated disproportionately to the temperature and other signs. Finally, it is evident that there is a gastrointestinal disturbance, but the manifestations of this are not constant. Difficulty in moving the bowels without complete obstruction or, less commonly, with bouts of diarrhea, is seen in nearly all cases. Vomiting is variable. Bloody diarrhea should not be expected, but when found, it is a valuable contributory sign. Rapidly progressing shock is uncommon and in our experience is more frequently seen in extensive venous occlusion that in arterial occlusion.

We have been able to establish no criteria by which we can distinguish between venous and arterial occlusion other than a fortuitously obvious etiologic factor. Some observers have noted that arterial thrombosis is more rapid in onset with marked toxicity and is rapidly and progressively fatal. This is not invariably the case. In fact, in our experience the most rapidly fatal case was one of extensive thrombosis of the mesenteric veins. Moreover, the experimental observations of Scott and Wangensteen[11] indicate that a venous occlusion, if extensive, should be rapidly fatal because there is a tremendous loss of blood from the arteries into the involved sement of bowel. Warren and Eberhard[5] also have stated that the differential diagnosis between arterial and venous thrombosis is highly speculative.

Hibbard[12] has called attention to the roentgenographic manifestations of experimental mesenteric vascular occlusion. He found that moderately distended loops of bowel, gas shadows, and fluid levels could be detected by means of a plain x-ray film taken in the upright position a few hours after the experimental ligation of the mesenteric artery or vein in dogs. These signs developed before there was clinical evidence of obstruction. Although not distinguishing between vascular occlusion and mechanical intestinal obstruction, he felt that this evidence would be of value in establishing an early diagnosis. However, it should be pointed out that while such evidence may be of assistance, a negative Roentgen examination of the bowel is of no diagnostic value. In the present series seven patients had had x-ray studies which varied from a plain film of the abdomen to a complete examination of the gastrointestinal tract by means of a barium enema and barium meal. In none of these cases was there conclusive evidence of intestinal obstruction. Consequently, the x-ray should

be considered only as a means of furnishing contributory evidence and is not in any sense conclusive.

It should not be maintained that the features which we have outlined are pathognomonic of mesenteric vascular occlusion nor are these essential to diagnosis, for the white blood count, the temperature, and the physical findings may be as misleading in this condition as in appendicitis. However, these features, when present, render a diagnosis much more tenable than is generally realized. The condition which we have had the most difficulty in distinguishing from mesenteric vascular occlusion is a subacute pancreatitis in an elderly patient. However, the temperature tends to be higher in pancreatitis, vomiting is a more prominent symptom and the pain in subacute cases is more easily relieved by sedatives. In the severe cases of pancreatitis, signs of early peritonitis are more striking than in mesenteric vascular occlusion. The blood diastase is of value in the differential diagnosis of doubtful cases.

It may be argued that all cases do not conform to the syndrome which we have outlined. Every clinician of experience can recall cases in which an entirely unsuspected vascular occlusion of the mesentery was revealed at post-mortem. The reason for this lies not in the site, type or degree of the occlusion, but in the underlying etiologic factors or intercurrent disease. Thus, in 30 per cent of the arterial cases in the Peter Bent Brigham Hospital series the presence of extensive coronary artery disease or cerebral arteriosclerosis had rendered the patients moribund before the abdominal vascular accident occurred. Similarly in 60 per cent of the venous group the terminal stages of cirrhosis of the liver or intra-abdominal sepsis were associated with an unrecognizable thrombosis of the mesenteric veins. These cases, however, in no way detract from the value of the syndrome described above in the recognition of early mesenteric vascular occlusion. Given a patient with (1) severe abdominal pain; (2) a paucity of physical findings so that the picture as a whole is confusing and

does not fit any of the usual surgical emergencies; (3) a high leucocytosis and high pulse rate with a relatively low temperature; (4) abdominal tenderness more or less generalized; and (5) evidence of a gastrointestinal disturbance manifested by either vomiting or diarrhea or signs of subacute obstruction—mesenteric vascular occlusion should be considered as the most likely cause.

It is not generally realized that one of the rarer manifestations of abdominal vascular disease is intraperitoneal hemorrhage. This condition is the abdominal counterpart of cerebral hemorrhage. Less than a dozen cases have been reported in the literature. Although very rare, it is of sufficient interest to warrant a brief description. The clinical picture is one of severe abdominal pain accompanied by signs of peritoneal irritation, shock and hemorrhage. In the majority of the reported cases the diagnosis has been acute pancreatitis or perforated ulcer. In the only case which we personally have observed[13] the clinical picture was quite suggestive of mesenteric vascular occlusion which was the preoperative diagnosis.

A word must be said in regard to the indications for operative intervention in abdominal vascular disease because, as has been emphasized before, spontaneous recovery may ensue. Actually in the majority of cases by the time the diagnosis has been established, operation is imperative. However, with an increasing interest in the disease, cases are being recognized earlier and there is such a thing as operating too soon, because (1) the disease may not progress to gangrene and (2) even if gangrene is imminent a very early operation may find the lines of demarcation poorly defined. This situation is particularly likely to arise in the case of a patient with auricular fibrillation already in the hospital for medical care. Following a sudden change from fibrillation to a normal rhythm the patient may show evidence of a small embolus to the brain or extremity and then suddenly present the picture of an abdominal castastrophe. There may be severe abdominal pain, vomiting, and

shock of considerable degree. The diagnosis of a vascular accident in the abdomen is obvious and the tendency is to operate at once. We feel that in such cases one should treat the patient for shock for a period of at least several hours, noting at frequent intervals the blood pressure, pulse rate, leucocyte count and temperature. Careful repeated examinations of the abdomen are essential. A steadily rising pulse rate and leucocyte count and increasing abdominal tenderness, even without evidence of marked rigidity or abdominal distention, are indications for surgery. If, however, the white count rises only slightly, the pulse rate tends to stabilize and no abnormal physical findings are noted, a conservative policy is indicated and recovery may be expected.

SURGICAL MANAGEMENT

Once operative intervention is elected there is no place for conservative measures in the treatment of abdominal vascular disease. Enterostomy, sometimes a life-saving procedure in intestinal obstruction, is worthless once gangrene has been established. Exteriorization is also fraught with danger because if the thrombosis is venous, there is a tremendous loss of blood into the involved segment of bowel. Moreover, even in arterial occlusion the removal of the gangrenous segment of bowel is accompanied by a striking immediate improvement in the patient's condition, as if there had been absorption of toxic products from the gangrenous loop.

As has been emphasized many times before, the poor general condition of these patients is no argument against resection since the alternative is a fatal issue. There is but little trauma to a well conducted resection and it is amazing how well these patients survive an apparently hopeless situation. Thus, recovery has ensued in a case in which no anesthesia was necessary because of the moribund state of the patient[14] and in nearly every case the patients have seemed to be in better condition immediately after than immediately

before the operation. This striking improvement is undoubtedly directly related to the complete removal of the gangrenous bowel.

Although the necessity for resection is well established, the proper treatment of the divided loops of bowel is a debatable point. An immediate anastomosis, as recommended by Elliot,[15] is obviously the ideal procedure. However, in his successful case Elliot performed a secondary closure. Jackson, Porter, and Quinby[16] cautioned against immediate suture because of the difficulty in determining lines of demarcation and the danger of advancing gangrene. More recently a multiple stage method has been advised by Green and Allen.[17] It seems to us, however, that each case must be handled as an individual problem. If the lines of demarcation are well defined and the condition of the patient is satisfactory a primary anastomosis can be done. On the other hand, exteriorization of the divided loops may be the procedure of choice in the face of poorly demarcated necrosis or a badly reacting patient. It is worth noting that in a study of the reported operative cases of the fourteen years between 1920 and 1934 the highest percentage of recoveries was found to occur in the group in which resection with immediate anastomosis was done.[2]

In the operative approach a generous right rectus incision should be used and the cecum and terminal ileum at once identified. The extent of involvement of the colon should be determined and then the small bowel explored proximally, delivering the involved segments at the same time. When the proximal border of the gangrene is found, the entire extent of the lesion is known and the resection can at once be begun. If the terminal ileum is not involved, exploration from this point proximally should be done as the colon and upper jejunum are rarely involved. If the exploration is systematically done in this order very little time is lost in determining the extent of the lesion and a good start towards a well conducted resection is obtained.

There are several points to be noted in the surgical management of intra-abdominal apoplexy. If the diagnosis is suspected a rather high right rectus incision should be used. It is more likely, however, that the surgeon will encounter an abdomen full of blood in an elderly individual in whom the diagnosis has not been suspected. In such cases the gastrohepatic ligament should be investigated first as it is the most frequent source of hemorrhage. If the ligament is swollen and thickened by active hemorrhage, it should be opened and the bleeding vessel identified and ligated.[18] However, if all signs of continued hemorrhage have ceased, it may be permissible to leave the area undisturbed without identifying and ligating the precise bleeding point.[13] If the gastrohepatic ligament is not involved and the gastric vessels appear normal along the greater curvature, the superior mesenteric artery should be investigated. This is the next most common site for intraperitoneal hemorrhage in abdominal vascular disease.

SUMMARY

1. The etiologic factors involved in mesenteric vascular disease are not sufficiently obvious clinically to be considered essential in the diagnosis of this condition.

2. In the majority of cases the gross pathologic anatomy of arterial and venous mesenteric vascular occlusions is quite similar. The pathogenesis of each is such that the onset of the disease may be sudden or slowly progressive. Consequently, except for a fortuitously obvious etiologic factor, no reliable distinguishing features should be expected clinically.

3. The clinical manifestations of mesenteric vascular disease may be divided into three groups: those which result from spasm or small, non-fatal occlusions of the mesenteric arteries, so-called "abdominal angina" or "abdominal intermittent claudications"; those which follow an extensive occlusion of either a mesenteric artery or vein, "mesenteric vascular occlusion"; and those which are a consequence of the rupture of a sclerotic artery with intra-

peritoneal hemorrhage, "intra-abdominal apoplexy."

4. The clinical characteristics of each of these three groups have been discussed in detail and certain points in the surgical management of these cases have been outlined.

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INTERPOSITION OF VITALLIUM PLATES IN ARTHROPLASTIES OF THE KNEE

PRELIMINARY REPORT

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THE reconstruction of a new joint by arthroplasty is a complicated surgical procedure involving the consideration surfaces of an ankylosed joint is by no means a new procedure, having been tried and abandoned over fifty years ago.
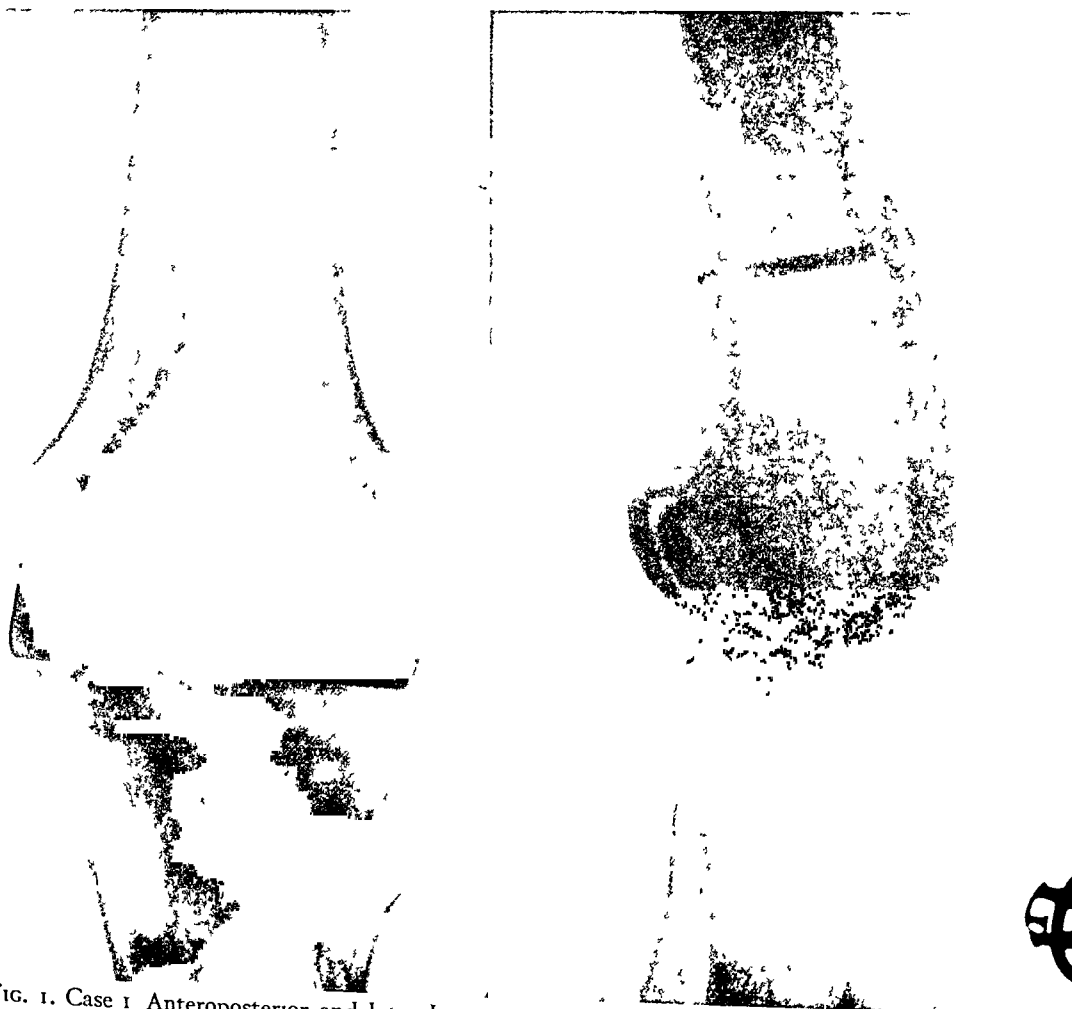
FIG. 1. Case 1 Anteroposterior and lateral roentgenograms of knee following arthroplasty, showing vitallium plate in position.

of many factors. The interposition of fascia lata or other material as a lining of the new joint is only one of several important steps in the operative technique.

The interposition of various metals, as gold and silver, between the articular Smith-Petersen has revived this principle in arthroplasty of the hip joint, employing an entirely different method whereby a non-irritating metal cap is placed over the head of the femur. He first tried various materials for this purpose, as glass and

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bakelite, but has recently adopted the use of caps made of the alloy vitallium, which was introduced by Dr. Charles Venable.

brane, reported in the Robert Jones Lecture in 1930. In Smith-Petersen's cases, however, a longer period of time had
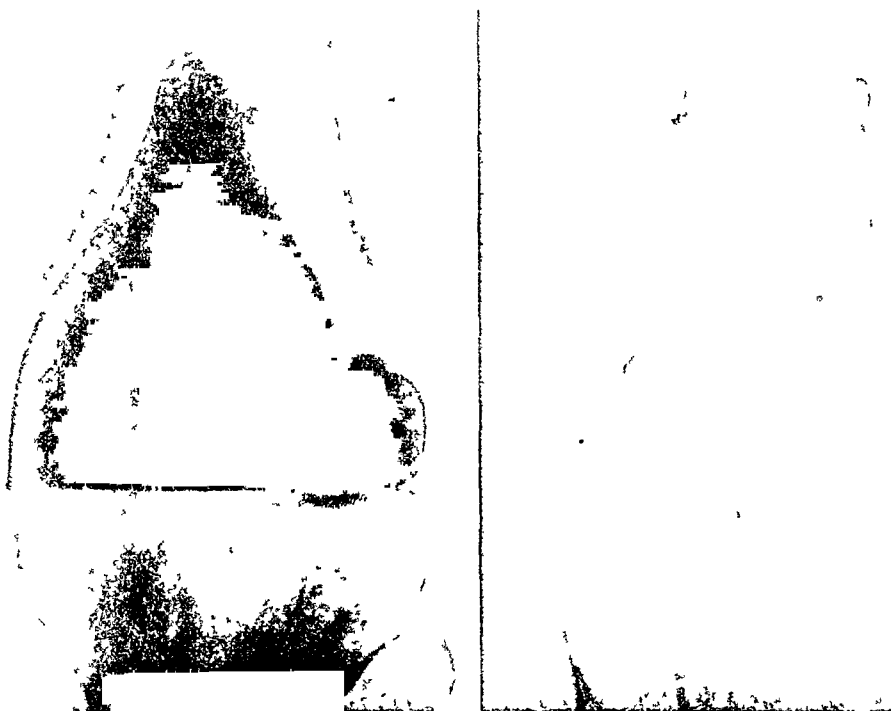
FIG. 2 Case II. Anteroposterior and lateral roentgenograms of knee following arthroplasty, showing vitallium plate in position.

The cap is not fixed in any way to the bone; thus, free play is possible between the cap and the head of the femur, as well as between the cap and the acetabulum. He has employed this method in a small number of cases of bilateral ankylosis as a result of atrophic or rheumatoid arthritis, with excellent results. The procedure was also carried out in one case of ankylosis following a pyogenic infection; sufficient time had not elapsed since this operation to determine the end result.

In one patient, Smith-Petersen demonstrated a fibrocartilaginous lining in the new joint following the interposition of the vitallium cap, similar to the evolutionary physiologic changes observed by the author after the use of fascia lata as a lining mem-

elapsed and the evolutionary process had reached a more mature stage at the time of operation than in those of the author; further, a smoother and more normal contour of the interior of the joint was obtained than in the author's cases, wherein fascia lata had been interposed.

Venable has also employed a vitallium cap for lining the joint in arthroplasty of the hip. He fixed the cap by a metal screw, allowing motion only between the cap and acetabulum.

These measures have not been tried by a sufficient number of surgeons, nor has a proper length of time elapsed to permit an estimation of their true value, but the results shown by Smith-Petersen warrant further investigation. With this in mind,

the author has interposed a vitallium plate in two patients with ankylosis of the knee following acute pyogenic infectious arthritis. The technique of the operation is similar to that described by the author for arthroplasty of the knee where in fascia lata is used, although slightly less joint space was created than has been customary for the interposition of the membrane. Prior to the operation, the size of the plate was estimated from roentgenograms and constructed to fit over the anterior surface of the lower end of the affected femur. Since the anatomy of the knee joint precludes the interposition of metal without internal fixation, the plate was maintained in position by two posterior triangular flanges hooked into the posterior surface of the condyles, and by one vitallium screw inserted into the anterior surface of the shaft of the femur.

In the two cases in which this plate has been interposed, we have been disappointed in the amount of motion obtained. This may perhaps be due to an inadequate joint space. In the future, we plan to enlarge the joint space for interposition of vitallium to equal that of joints which are lined with fascia lata. The proper joint space between the articular surfaces of the femur and tibia is approximately ¾ inch.

We are now working on a modification of these plates in order to obtain one which can be more easily removed, if necessary. We are also attempting to model a plate to fit the upper end of the tibia, to be used instead of the plate on the lower end of the femur.

Our results following arthroplasty of the knee wherein fascia lata was interposed have been so satisfactory that this material will by no means be discarded until some other substance can be secured which will minimize the operative procedure and provide an even more normal joint. In science, one should possess an open mind, and if the results can be improved by new methods, they should be adopted. It is in this spirit that we are now using vitallium plates in arthroplasty of the knee. A further report will be made after the trial of this metal in a larger number of cases.*

CONCLUSIONS

The use of vitallium in the reconstruction of a new joint is in the experimental stage, and the ultimate outcome of these cases is at present doubtful. Smith-Petersen's results in the hip have been encouraging and have led the author to try this method in the knee joint. Sufficient length of time has not elapsed, however, to determine whether or not the interposition of vitallium in the knee will be of permanent value, as the problems connected with arthroplasties in this joint differ materially from those in the hip. A further report will be made on these cases when sufficient time has elapsed to determine the end results.

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* Since the submission of this paper for publication, two additional arthroplasties of the knee have been performed wherein vitallium was interposed.

DISCOGENETIC DISEASE

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I

IN 1824, Wenzel[25] described a disease "in which the intervertebral discs are involved early and almost exclusively, whereby we find at the vertebral bodies a just beginning morbid change which consists in inflammation." About twenty years later, Rokitansky[17] stated that the common type of spondylitis deformans (which is marked by exostoses at the margins of the vertebral bodies) is a sequel of degenerative thinning of the intervertebral discs. Half a century later, and within one year after the discovery of the x-rays, Beneke[1] reported the results of his autoptic and roentgenographic studies in an almost inaccessible publication. Confirming Wenzel's and Rokitansky's views on the pathogenesis of spondylitis deformans, he added that static factors are essential in the production of this disease which he grouped with osteoarthritis.

This, as well as Léri's[10] Eugen Fränkel's[5] and Simonds'[21] discerning work on the subject, was almost forgotten when, about twelve years ago, Schmorl and his pupils[19] rediscovered the pathogenetic significance of lesions of the discs in diseases of the vertebral column, especially in the common disease which they termed "spondylosis deformans" (hypertrophic or osteoarthritic spondylitis of the Anglo-American literature). Their observations have since been confirmed by many authors. Experimentally, this type of spondylitis was produced by injury to the discs in dogs,[8] and, by accident, in man.[11]

According to these investigations, the pathogenesis is as follows: Thinning of the discs induces, first, abnormal mobility of the adjacent vertebral bodies, thus causing abnormal stress upon the ligaments that connect them; secondly, abnormal contact between adjacent vertebral surfaces; finally, abnormal position of the articular processes. The stressed ligaments may calcify; contact between vertebral bodies may produce condensation and exostoses; in the displaced facets of the intervertebral (apophyseal) articulations, osteoarthritis may develop; and the diminution of the distance between adjacent vertebrae, together with the displacement of articular processes, may considerably narrow the intervertebral foramina, thus causing pressure upon the nerves that pass through them. Thinning of the discs may be due either to trauma, or to a complex degenerative process, or both; irrespective of its origin, however, it is this thinning which underlies all the changes above mentioned—these changes taking place in vertebral structures that are not primarily diseased. On the other hand, infection or new growth in the vertebrae may secondarily involve the discs; but the mechanical alterations act then upon diseased areas, e.g., softened bone; the results, differing entirely from those observed in normal vertebrae,[13] do not form part of this discussion.

II

Hypertrophic spondylitis may develop within a few months after the discs have been injured;[11,16] but, in other instances, no noticeable changes occur in the vertebrae during indefinite periods of observation. Beneke reported one such case; he believed that, since the patient had been confined to bed for several years, exostoses failed to develop because the normal mechanical stress upon the spine had ceased; and he concluded that persistence of this stress after the discs thinned down is essential in the production of hypertrophic

spondylitis. The following examples may illustrate the fact that other factors also are involved.

A healthy woman incurred a severe trauma of the spine at about 40 years of age. Ten years later, radicular neuritis with wasting of the muscles of one upper extremity had gradually developed and lasted for the subsequent twenty years. About forty years after the accident, when neuritic symptoms recurred, roentgenograms showed obliteration of the fifth and sixth cervical intervertebral spaces, indicative of thinning of their discs, but practically no deformities or condensation of the adjacent vertebrae. Three years later, the aspect of the spine had not changed. (Fig. 1.) Throughout these forty years, however, the patient had led a very active life.

For comparison, Figure 2 shows roentgenograms, taken at an interval of eleven months, of a woman of about the same age and build who also suffered from severe radicular neuritis: the rapid progress of condensation and exostoses during this interval contrasts strongly with the previous instance.

In other cases, some of the vertebrae adjacent to thinned discs form large exostoses, while others remain normal. (Fig. 3.) These observations suggest that persistence of normal stress upon the spine is in itself not sufficient to account for the production of bone hypertrophy; the readiness of the bone to respond to irritation seems to vary in different persons, as well as in different sections of the selfsame vertebral column.

Vertebral exostoses commonly originate at the junction of the annular epiphysis (Schmorl's "Randleiste") with the main part of the vertebral body.[19] This is also the place where the anterior longitudinal ligaments represent to some extent the periosteum of the vertebral bodies.[19] When they are under stress, irritation may lead to "periosteal" ossification as in other bones; this is said to occur when the discs, growing soft, protrude beyond the margins of the vertebrae. Eburnation and exostoses may also develop at the horizontal surfaces of the vertebrae, when these come in contact because the discs thin down. Similar exostoses and ligamentous ossifications,

however, may occur when the discs are not diseased, e.g., in infectious spondylitis; and well marked calcification of longi-

A   B
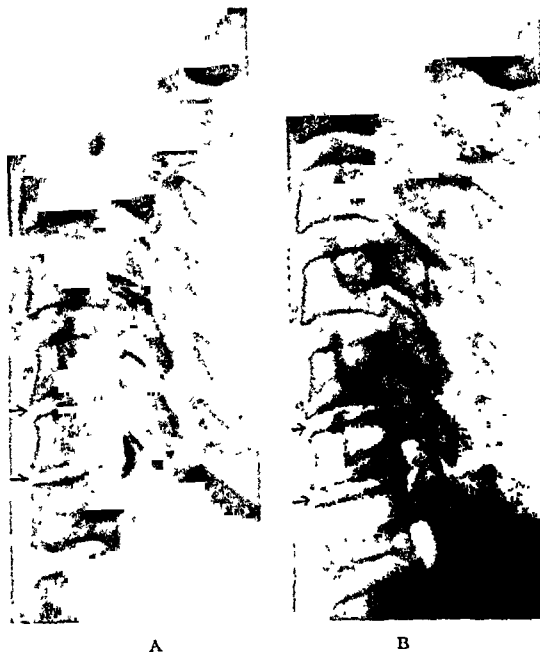
Fig. 1. A, the fifth and sixth cervical intervertebral spaces are narrowed. No exostoses. Apophyseal joints free. B, three years later, the condition is unchanged. Case referred to in text.

tudinal ligaments may, for reasons yet unknown, occur in vertebral sections that are otherwise normal, especially in sections of reduced mobility.[14] The ligaments ossify quite commonly in spondylose rhizomélique (spondylarthritis ankylopoietica; Struempell-Marie disease; falsely termed Bechterew disease in the German literature), chiefly after the intervertebral apophyseal articulations have undergone bony ankylosis.[14] In this disease, usually the ligamenta flava are more severely affected than the other ligaments,[10] but recent reports show that thickening and ossification of the ligamenta flava may also occur as a disease independent of other spinal lesions.[12,14,23]

When, as a result of thinning of the discs, the distance between adjacent vertebrae diminishes, their articular processes obviously become displaced. In the cervical spine, where the plane of the facets slopes diagonally backward and downward, the superjacent articular process will slide backward along the subjacent facet; in the

dorsal and lumbar spine, where the plane of the facets is more vertical, the superjacent vertebra slides downward rather

hypertrophic spondylitis with normal apophyseal articulations in the cervical region, but definite hypertrophic spondylarthritis
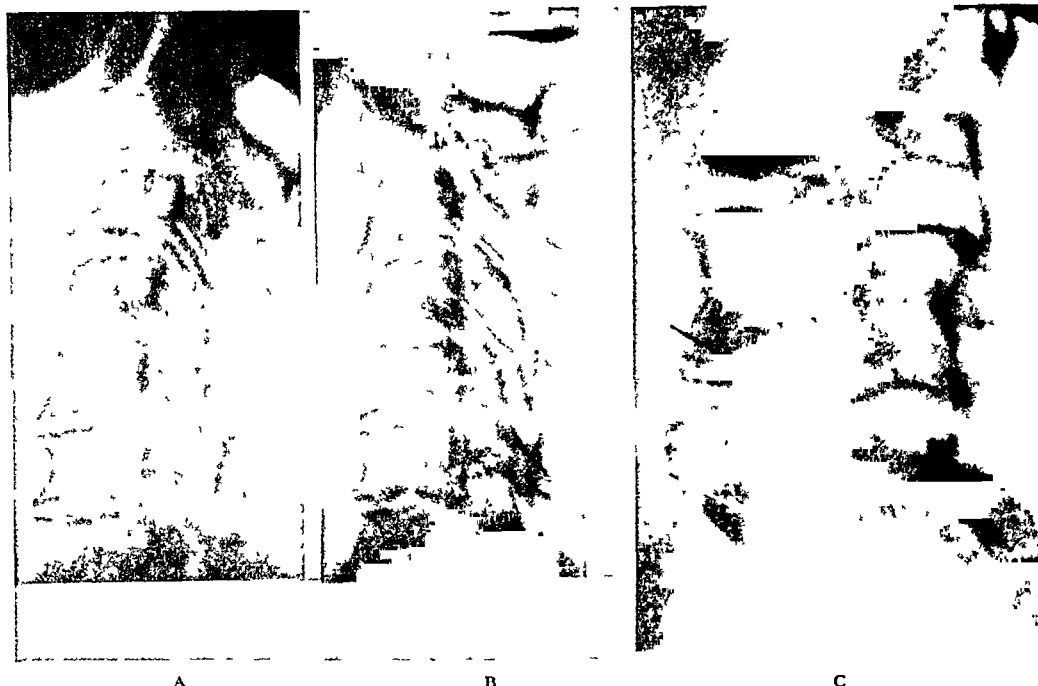
FIG. 2. A, cervical spine. Thinning of discs with hypertrophic spondylitis. B, eleven months later. Note progress of exostoses. Apophyseal joints free. C, the lumbar spine of the same patient. The two lower discs are thinned, the articular processes are displaced (arrows), their bony density is increased, and the facets are ragged. However, there are no exostoses at the vertebral bodies. (Hypertrophic spondylitis in the cervical spine, hypertophic spondylarthritis in the lumber region, both due to thinning of discs.)

than backward. This, together with the greater weight upon the lumbar region, exposes the tips of the articular processes in the lumbar spine to greater stress than in the cervical, when the discs thin down. Osteoarthritis of the intervertebral articulations may develop (hypertrophic spondylarthritis) but it is noteworthy that it fails to do so in the majority of cases.[14]

This behavior is similar to that observed in postural anomalies, e.g., scoliosis, where the joints under stress do not of necessity become diseased. Apparently the variable resistance of the articular cartilage of the apophyseal joints is responsible for these differences. This is a factor distinct from the variable readiness of the bone to form exostoses, for hypertrophic spondylitis and hypertrophic spondylarthritis are not interdependent. Figures 2 and 4 show in conjunction with thinning of discs marked

with normal vertebral bodies in the lumbar spine.

Like hypertrophic spondylitis, hypertrophic spondylarthritis occurs also in the absence of any changes suggesting abnormal stress, e.g., in persons otherwise healthy who are infected with entameba histolytica.[14]

It would seem, then, that in thinning of the intervertebral discs, the vertebral bodies, articular processes, and intervertebral ligaments, while invariably exposed to abnormal mechanical stress, do not of necessity develop hypertrophic spondylitis and hypertrophic spondylarthritis; whereas, on the other hand, hypertrophic spondylitis and spondylarthritis may and do occur in the presence of normal discs. This suggests that spondylitis and spondylarthritis of this type are merely responses to various forms of persistent

irritation, either from without (mechanical), or from within (infectious, toxic). Hence, thinning of discs, being only one of the causative factors involved, should not be considered identical with the vertebral morbid changes that it may or may not induce.

III

Broadly speaking, the discs may thin down in any region of the column, for example, when they are affected by injury, infection, or new growth. But in the more common gradual thinning which is called degenerative, two typical localizations are observed. First, in a rare form of kyphosis, the anterior halves of the discs in the midthoracic spine are flattened by the increasing pressure of the vertebrae upon them. Anatomically, this condition corresponds to Schmorl's "Alterskyphose," and, to some extent, to Knaggs' "spondylosis muscularis," possibly also to the one case autopsied by Bechterew. Junghanns pointed out that this type of kyphosis could not develop unless the vertebral bodies of the affected region were comparatively normal, and that softening of vertebrae may be associated with thinning of the discs in some instances. Previous studies gave me the impression that the type of kyphosis here discussed is not a distinct entity, but a more or less accidental occurrence; for in the pathology of the spine much more depends on the reciprocal hardness and elasticity of discs and vertebral bodies than on the absolute resistance of these parts.

Secondly, in the vast majority of cases, the discs become thinned in the lower cervical and lower lumbar regions. (Figs. 2 and 4.) These regions, being the areas of greatest mobility, are supposedly exposed to the greatest mechanical stress under physiologic conditions.

This does not agree, however, with the fact that when the vertebrae are softened, they usually flatten or collapse in the midthoracic spine and, less commonly, in the upper lumbar region, while they remain

normal in height at the cervicodorsal and lumbosacral junctions (where the discs are thinned so commonly). This is illustrated

FIG. 3. Hypertrophic spondylitis in thinning of discs. All the lumbar discs are thinned down (x), but not all the vertebrae form exostoses (arrows). Apophyseal joints perfectly normal. (Spondylitis without spondylarthritis.)

in Schmorl-Junghanns' book, in which most of the roentgenograms and specimens demonstrate that the midthoracic vertebrae are flattened whenever there is systemic weakening of vertebral bodies. Similarly, in eleven cases of non-localized injury to the spine (fall from a height of more than 6 feet), we found only the midthoracic vertebrae fractured and com-

pressed. This corresponds to the observation that in multiple vertebral fractures the midthoracic vertebrae are those that

IV

As a rule, the clinical symptoms produced by "chronic degenerative" thinning
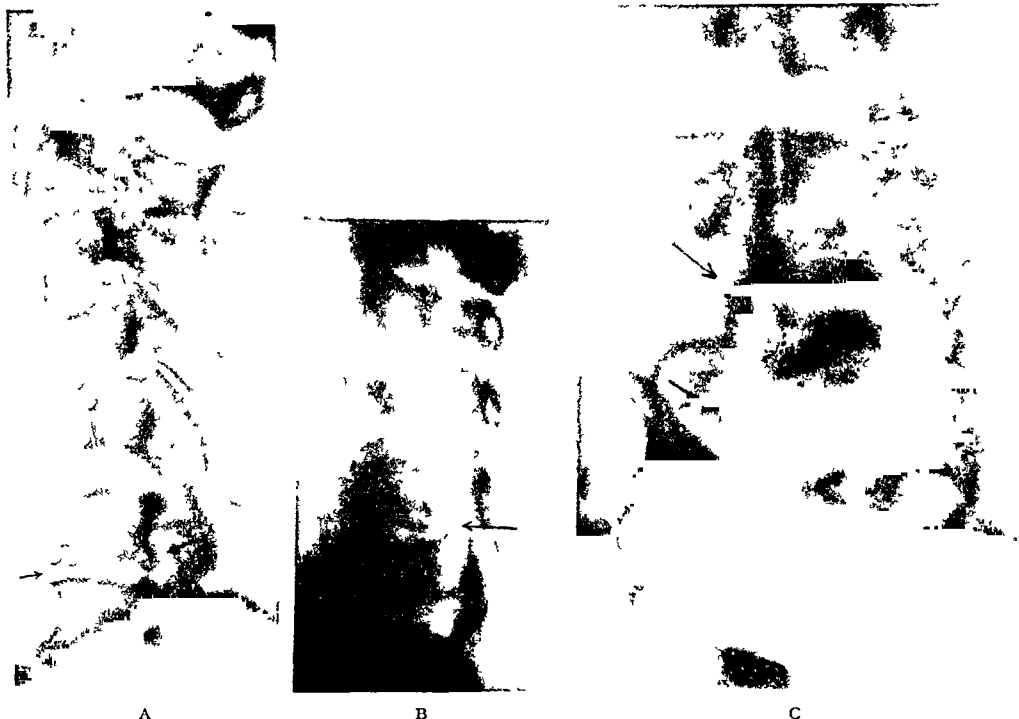
FIG. 4. A and B, typical localization of "degenerative" thinning of discs in the cervical spine. C, same in lower lumbar spine. Hypertrophic spondylitis in the certical spine, with exostoses at the posterior borders of the vertebral bodies, bulging into one intervertebral foramen (arrows) Hypertrophic spondylarthritis in the lumbar spine, with displacement of articular processes and narrowing of the apophyseal joint spaces (arrows), in the presence of normal vertebral bodies. See text.

are most commonly involved.[2] In two of our cases, one disc in the lower cervical spine was injured simultaneously with midthoracic vertebrae. The midthoracic vertebrae are also found collapsed in a peculiar type of senile osteoporosis, where the condition may actually amount to a reversed picture of that usually encountered in thinning of discs.[13]

It is difficult to see why the physiologic mechanical stress should be most marked in the midthoracic spine when the vertebrae grow soft, but most pronounced in the lower cervical and lumbosacral regions when the discs lose their elasticity. It seems, however, as though softened vertebrae tend to collapse in the region of physiologic kyphosis, whereas, other things being equal, the discs tend to thin down in the regions of natural lordosis.

of intervertebral discs are quite distinctive. Owing to the common localization in the lower cervical and lower lumbar regions, where the nerves that pass through the intervertebral foramina are especially thick, radicular neuritis is a frequent manifestation. Since the displacement of vertebral structures which produces narrowing of these foramina naturally precedes the formation of any reactive changes in vertebral bodies, ligaments and, especially, in the apophyseal articulations, irradiating pain in the extremities usually precedes the onset of local pain in the back by many years. This "referred" pain is not easily recognized as being of nervous origin, because objective signs of radicular neuritis do not, as a rule, develop at early stages of root compression. Clinically, the neurogenous nature of this "rheumatism" does not

often become evident until wasting of muscles or trophic disorders prove it.[16] Local pain is more common in the lumbo-

Fig. 4D. Thoracic spine normal.

sacral than in the cervical region; it is usually entirely absent in the latter. This agrees with the observation above reported, that hypertrophic spondylarthritis as a result of thinning of discs is more common in the lumbar than in the cervical section.

The clinical course of the disease is usually marked by three conspicuous features. First, the symptoms are not persistent, but recurrent; secondly, they vary in character at different periods;

thirdly, they are not proportional to the degree and amount of anatomic changes as found roentgenographically.

Fig. 5. Exostoses at the posterior borders of the vertebral bodies bulge into the intervertebral foramina (arrows). Apophyseal joints free. The case illustrates that minute changes, which escape recognition in conventional roentgenograms, may by their localization give rise to compression of nerve roots.

The clinical signs and symptoms have been described in previous reports.[24] It may here be mentioned that among the 261 patients observed, the majority sought medical advice at about 50 years of age, although the history usually reached back to earlier periods, often even to adolescence. "Rheumatism" or "sciatica" of a few days' duration recurred at intervals of from eight to twenty months. After a number of years the trouble increased in severity, duration, or frequency, until it sometimes amounted to disability of the affected limb. This stage usually lasted for two or three years, after which time the symptoms tended to subside or disappear, although acute exacerbations occurred occasionally. One may finally find the intervertebral spaces entirely obliterated or, in other cases, the vertebrae adjacent to narrowed interspaces fixed by fusion of contiguous spondylitic exostoses; in either case, there are often no more symptoms.

As in other types of spondylitis, riding a car or taking a long walk often brings on the pain; but it is almost pathognomonic of thinning of discs that the pain does not subside during rest at night (as it does in inflammatory diseases of the vertebrae and intervertebral joints); on the contrary, pain after midnight is a very common complaint.

According to these observations, it would seem that weakened discs thin down at intervals, possibly after slight traumas such as may be incurred in daily life. Schmorl found in degenerating discs small fissures at their margins and attributed these lesions to repeated slight traumata. He considered them responsible for leakage of disc material. Correspondingly, one may find under fortunate circumstances that the intervertebral spaces remain of constant width for a period of years, but diminish rather suddenly at irregular intervals. (Fig. 3.) By the narrowing of the intervertebral foramina thus produced, as well as by leakage of the gelatinous disc material backward, compression of nerves may then occur where they are thick. Since the leaking material gradually dries out, the original amount of compression diminishes after a certain period, until a new fissure brings on another recurrence. Since the lordotic position narrows the foramina,[3] movements and positions associated with lordosis commonly accentuate the discomfort. In the lumbar spine this results from backward stretching, and in the cervical by the supine position during rest. When actual herniation of the discs occurs into the spinal canal, the compression becomes permanent in many cases.

The treatment consists in the prevention of any injury to the spine. Avoiding sudden movements, wearing rubber soles and heels, supporting the neck at night by the addition of pillows, are often very helpful measures. In acute exacerbations, ultrashort wave therapy is the most effective treatment known at present. In radicular neuritis associated with trophic disorders of the skin, muscles or bones, one is impressed with the rapid recovery under this treatment of tissues that seemed to be very seriously damaged.[16] In other less dramatic cases a second recurrence often fails to respond to this treatment as favorably as the first.

Irritation rather than relief may be caused by too frequent or too strong short wave applications. Three treatments a week, of fifteen minutes each, given during a fortnight, are usually sufficient. The patient should not experience any sensation during the treatment.

We have now given up manipulations and massage, since they often tend to increase pain. Exceptionally, when short wave treatment is not successful, Roentgen therapy over the diseased area may prove to be helpful (100 r, 155 kV, 4 ma., 0.5 mm. Cu; at intervals of about one week).

V

It has often been observed that compression fractures of vertebral bodies may remain unnoticed by the patient; and it is well known that destruction of vertebral bodies, e.g., in Pott's disease, may cause no symptoms for a long time. According to a recent report,[6] tuberculosis of the intervertebral discs causes severe radicular neuritis. While large exostoses at the anterior borders of the vertebrae are often accidentally discovered in the absence of symptoms referred to the back or limbs, small exostoses at the posterior borders may be responsible for severe segmental neuritis[16] when they bulge into the foramina. (Figs. 4 and 5.)

These findings may throw some light on the much debated question of the actual clinical significance of hypertrophic spondylitic changes. Neither the presence nor the absence, nor the amount of bone changes determines the severity of the clinical symptoms. The degree of thinning of the discs is by no means proportional to the discomfort experienced by the patient. Both the vertebral bodies and the articular processes may become immobilized by fusion of exostoses, in which case pain

ceases although the bone changes are very pronounced. The clinical syndrome associated with spondylitis and spondylarthritis is no more dependent on the amount of "spiculations" or deformities than, for example, the clinical course of pulmonary tuberculosis depends on the amount of conspicuous shadows found in the roentgenogram of the chest.

By the addition in recent years of a number of new facts to the pathology of the spinal column, our conception of spinal disease is being altered at present, and the classifications in use are no longer adequate. The large number of terms and the lack of conformity in their interpretation seem to indicate that these classifications have ceased to be entirely satisfactory. For instance, the disease here discussed is variously termed spondylosis muscularis, spondylitis or spondylosis deformans, trophostatic osteoarthrosis, hypertrophic spondylitis, polyspondylitis marginalis osteophytica, arthritis of the spine, and Bechterew's disease—the two latter terms being also used to designate spondylarthritis ankylopoietica, a condition which has almost nothing in common with the former.

It does not seem justifiable to add another name to this confusing variety, unless this name serves to define a distinct entity and to eliminate terms the meaning of which has become incorrect, dubious, or merely dogmatic. Since in the disease here under discussion a group of anatomic alterations in vertebral bodies, intervertebral articulations, and ligaments, as well as a distinct though variable clinical syndrome, originate in a disease primarily affecting the discs, the term "discogenetic disease," suggested in previous reports, is here adopted as a tentative abbrevation. It may serve to define a common clinical and anatomic syndrome and to distinguish it from the type of spondylitis and spondylarthritis with which it may or may not be associated, but with which it is not identical. To characterize these vertebral changes more fully, one may speak of the discogenetic form of spondylitis and spondylarthritis.

SUMMARY

There is no constant interrelation between thinning of the intervertebral discs and hypertrophic spondylitis and spondylarthritis. The production of clinical symptoms depends neither on these bony changes nor on the amount of thinning of the discs, but on the localization of the entire process and the stage of the disease. In degenerative thinning, the lower cervical and lower lumbar regions are most commonly involved, wherefore, under certain conditions discussed in some detail, a group of clinical symptoms occurs frequently as a result of this affection. The clinical course, roentgenologic aspect, and mode of treatment are described. Since spondylitis and spondylarthritis are neither characteristic of this disease nor responsible for all the clinical symptoms, it is suggested that this group of anatomic findings and clinical manifestations be designated by the term "discogenetic."

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FRACTURE of the pubic portion of the pelvic ring . . . is the most common form of fracture occurring in the pelvis, and is caused usually by a squeeze between two solid objects or a fall from a height, the patient striking on the hip. It is quite frequently associated with other fractures in the pelvis.

From—"Fractures" by Paul B. Magnuson, 3rd Edition (Lippincott).

SURGICAL MANAGEMENT IN CASES OF ADVANCED DISEASE OF THE BILIARY TRACT

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ONE in every ten adults has gallstones and at least one more has cholecystitis without stones. They form a vast army of potential candidates for the complications of disease of the biliary tract. These complications are so numerous and so easily acquired that the candidate is sure of election if he lives long enough. With this thought in mind, the physician who undertakes the management of these patients by non-surgical measures assumes a grave responsibility. Medical treatment undoubtedly will be often successful in the control of symptoms and thereby it will confer a false sense of security upon both patient and physician. In the column of the surgical dyspepsias, disease of the gall-bladder leads all the rest.

The symptoms of cholecystitis are the most irregular and capricious of any lesion within the abdomen. But, once the physician has had his suspicions aroused he should not be content until the gall-bladder has been convicted with reasonable certainty, or unequivocally cleared of all suspicion. One must not be misled by the cholecystogram. When it indicates disease of the gall-bladder by definite signs such as non-visualization or filling defects due to stones—then it is accurate in over 90 per cent of cases. However in similar cases, clinical diagnosis, after careful interpretation of the history and physical examination, is also accurate in 90 per cent of cases. A gall-bladder which visualizes and empties in the cholecystogram may be markedly diseased and even contain stones. In this connection it is well to remember three facts:

1. Filling of the gall-bladder with bile depends on a functioning sphincter of Oddi at the lower end of the common duct.

2. The diseased gall-bladder, except in certain rare, usually acute conditions, can and does concentrate bile as witness the thick viscid bile in the diseased gall-bladder and the usually intact often hyperplastic mucosa and submucosa.

3. The wall of the diseased gall-bladder commonly is much thickened, a condition which the microscope reveals to be caused by marked hypertrophy of the intrinsic muscle fibers. This certainly indicates that the contractile and emptying mechanism has been overworked, probably because of some impediment to the outflow of bile.

There is an increasing tendency today to temporize with gall-bladder disease by so-called gall-bladder drainage, by diet, etc. Some of this is due to the fact that the physician hesitates to advise operation until urgent complications appear; or because he has had the unpleasant experience of mortality or morbidity. It goes without saying that surgery of the biliary tract is not the work for the inexperienced though daring tyro. In proper hands the risk is a reasonable one—no greater than that of any other major operation. It is not fully appreciated that cholecystitis, when once established, is a going concern that may have exacerbations and remissions but always produces a steadily progressive destruction. Is it not a fact borne out by

operating room experience that in every patient with disease of the gall-bladder who comes to operation there is visible damage to the liver; inflammatory changes in the common duct (which I was able to show by microscopic examination); and too often a swollen pancreas due to lymphangitis or to interstitial changes? Although serious enough, these are mild complications compared with others which may follow procrastination and delay.

Mrs. A. R., aged 62 years, was admitted to St. Joseph's Hospital April 5, 1937 with the chief complaint of jaundice which had been present for two weeks. About eight weeks before admission she had an attack of biliary colic which was relieved by a hypodermic of morphine. She recovered completely, but about four weeks later she had another attack of pain, not as severe as the first, followed in about forty-eight hours by the appearance of jaundice which became more marked and persistent until her admission to the hospital. Her only other complaint was a dull burning feeling in the right upper abdomen.

She had suffered from attacks of indigestion for a number of years but never had severe pain. Otherwise her general health had always been good. Her usual weight was 250 pounds.

Physical examination revealed an obese female 5 feet 6 inches tall weighing 210 pounds. She had no pain or discomfort. There was a generalized jaundice. Examination of her heart and lungs revealed nothing abnormal. Her blood pressure was 110/60. The abdomen was fat, soft and presented no palpable masses and no area of tenderness. In other words except for the presence of jaundice the patient did not appear to be ill. Her appetite and sleep were normal.

During the following two weeks there was very little change in her condition except several slight episodes of epigastric pain with radiation to the right shoulder and the fact that the jaundice persisted.

Laboratory Tests. The urine showed a faint trace of albumin, a few granular casts and the presence of bile. The blood count showed a slight leucocytosis. The blood Wassermann was negative. The Van Denberg test made on a number of occasions showed an immediate direct reaction and the icterus index varied between a low of 110 on May 3, 1937 and a high

of 170 on April 21. The blood sugar was 87 and the blood urea nitrogen 15.

I first saw the patient sixteen days after her admission, at which time I believed she had a stone in the common duct, although, according to the color of the stools, a small amount of bile had been coming through for two days. According to the temperature chart there was no infection. I recommended taking another icterus index, with operation if this showed the same or a higher value.

The stools became normal in color, indicating that the obstruction was somewhat relieved and there was improvement in the patient's general well being. She had a good appetite, no abdominal pain or tenderness, and normal temperature. She was therefore allowed to go home on May 4, 1937 under the care of her family physician.

The x-ray findings were interesting. The day after admission a flat x-ray plate of the abdomen indicated that the gall-bladder, filled with milk of calcium, was slightly enlarged. Two days later the patient was given the Graham dye and the x-ray revealed exactly the same density in the region of the gall-bladder previously reported. There was no evidence of non-opaque stones.

The patient was readmitted May 24th, with jaundice still present but somewhat decreased. The icterus index at this time was 45. The blood count showed a mild secondary anemia.

Operation was advised and carried out under ether anesthesia on May 26, 1937. An upper right rectus incision was made and the stomach and duodenum were found normal. The liver was enlarged, of slaty color and with rounded edges. The head of the pancreas was uniformly enlarged but not indurated. The gall-bladder was pale, distended and filled with stones. The common duct was dilated to a diameter of 2 cm. and several stones could be palpated floating up and down in the duct. When it was opened, a gush of dark bile appeared and two stones were found each 1 cm. in diameter, round and non-faceted. A curved hemostat was passed down the duct and into the duodenum, and the common duct was again palpated throughout. No more stones could be discovered. A No. 20 French rubber catheter was sutured in place in the common duct with its end upward toward the liver. The opening in the duct closed around it. The gall-bladder was then removed in the usual manner, from below upward, and the liver fossa oversewed

with catgut. There was very little bleeding at any time and the field was dry at the end of the operation. Two cigarette drains were placed in the subhepatic fossa, below but not in contact with the common duct. The wound was closed in layers around this drainage. The patient was given 500 c.c. of blood intravenously by the citrate method at the conclusion of the operation and she left the table in good condition.

The postoperative course was unusually smooth. Bile drained freely from the tube in the common duct. The two cigarette drains were removed five days after the operation and the catheter in the common duct came away spontaneously on the fifteenth day. The wound healed by primary union. The stools were of normal color and the jaundice was almost cleared at the time the patient was discharged from the hospital on the twenty-seventh day.

I saw this patient on July 28, 1937 at her home. She was feeling entirely well, had been enjoying walks out-of-doors and had no complaints referable to her old trouble.

From the standpoint of diagnosis this patient's history is rather typical of stone in the common duct, that is, attacks of indigestion over a period of years with recent biliary colic followed by jaundice. The only missing symptom was fever, which of course is indicative of cholangitis and is nearly always present in cases of this nature. Its absence in this patient did not necessarily mean absence of infection along the common duct or in the smaller ducts within the liver. I believe that in all such conditions definite evidence of infection can be found, although it may not be present in sufficient degree to give rise to systemic manifestations.

It should not be forgotten that biliary colic followed by jaundice may be due to obstruction at the lower end of the common duct either by pancreatitis or by carcinoma of the head of the pancreas. In other words, painless obstructive jaundice is usually caused by pancreatic malignancy, but on the other hand malignancy of the pancreas may occur in a patient who has gallstones. The patient with obstructive jaundice, regardless of whether the symptoms point to stone or cancer, nevertheless should be given the benefit of exploratory operation. It is not always possible to be certain of the diagnosis even when the pancreas is in the hand. I have seen a number of patients who had been operated on by experienced surgeons where the diagnosis of carcinoma of the head of the pancreas was proved to be erroneous by subsequent events.

When should operation be done in the patient with obstructive jaundice? How long is it safe to wait? What are the dangers and how can they be minimized? These are practical questions. The answers will often determine success or failure. Conditions vary in the individual patient so that it is difficult to lay down rigid rules of procedure. Much depends upon surgical judgment.

In general it may be stated that the patient with jaundice does not present an emergency problem. There is time for detailed examination particularly to note whether the jaundice is increasing or decreasing. This can be accurately determined by the icterus index. From a practical standpoint the color of the stools is important because it indicates whether or not bile is entering the intestine. When there is evidence that jaundice is decreasing and bile flow is becoming reëstablished it is advisable to postpone operation because in the presence of complete biliary obstruction there is a profound disturbance of the function of the liver which makes any surgical procedure extremely hazardous. A waiting policy does no harm unless there is active infection of the cholangitis type. In this event, and in spite of increased risk, external drainage of bile should be established by operation. However, this must be carried out as a minimum procedure; nothing should be done of a corrective nature such as removal of the gall-bladder or investigation of any other lesion. These should be left to a later time when the patient has passed the acute crisis.

In all these conditions the surgeon's chief ally is the wonderful recuperative power of the liver. There are on record cases of complete biliary obstruction of several

months' duration which have been relieved by the proper surgical procedure. I saw a patient not long ago who had complete biliary obstruction for eighteen months. He was deeply jaundiced, the liver was markedly enlarged but he was up and about and feeling fairly well. Operation was not done and at autopsy the obstruction was found to be due to stones in the common duct.

The chief dangers of operation in these patients are failure of liver function, renal insufficiency and hemorrhage. These can be minimized to a large extent by special preoperative preparation which should include blood transfusion, high carbohydrate intake and calcium chloride and glucose intravenously. The tendency to bleed in these patients presents a difficult problem. Frequently it is the cause of death in spite of the most careful safeguards. Notwithstanding the guidance of laboratory tests and the use of preventive measures, we know from surgical experience that some patients have uncontrollable bleeding and there is no certain way by which this can be foretold or prevented.

Operative Technique. I use an upper right rectus incision of adequate size beginning at the xyphoid notch. The suspensory ligament of the liver is divided to aid exposure. Palpation of the common duct with a finger in the foramen of Winslow, examination of the gall-bladder and investigation of the head of the pancreas are carried out. The stomach, duodenum and hepatic flexure of the colon are held firmly to the medial side by gauze pads under the hand of an assistant whose fingers straddle the gastrohepatic ligament. A clamp on the fundus of the gall-bladder enables one to pull it down and out and this makes accessible the extrahepatic structures. The common duct is opened by a small longitudinal incision and is then explored by a probe or curved hemostat for free passage into the duodenum. Usually these maneuvers will dislodge stones and milk them up toward the opening in the duct, from which they can be removed. A rubber catheter with a small

eye cut in its side is placed in the duct with its end upward toward the liver. It is fastened in place by one suture and then the opening in the duct is closed around it.

Attention is now directed to the gall-bladder which in most cases I prefer to remove. First, the cystic artery and cystic duct are clamped in one bite of the hemostat after the neck of the gall-bladder is separated from its loose attachment to the liver. I always remove the gall-bladder from below upward and try to do this mostly by sharp dissection while clamping any visible vessels which appear along the liver attachment. Before the gall-bladder is completely removed the pedicle containing the cystic artery and cystic duct is doubly ligated with chromic catgut. The gall-bladder bed is oversewed with catgut in order to cover raw surface and to control oozing. A dry field is desirable but not always possible and in such instances I do not hesitate to place a narrow double strip of iodoform gauze against any oozing raw surface of the liver. A cigarette drain is placed in the subhepatic fossa in order to guide any leakage of bile to the outside. The wound is closed in layers around the drainage material. The end of the catheter is placed in a 6 ounce bottle which is fastened to the dressings.

The complications of disease of the biliary tract present some of the most bizarre features encountered anywhere in pathology. The history of this patient is of unusual interest.

A woman, 69 years of age, was admitted to the hospital December 2, 1936 complaining chiefly of abdominal pain associated with distention and absence of any bowel movement for four days. The onset was sudden following a dose of sal-hepatica. This was followed by colicky pains on the left side of the abdomen which persisted to the time of her entrance to the hospital.

The chief features on physical examination were distention of the abdomen, exaggerated peristaltic activity, but no areas of rigidity or tenderness. The blood count revealed a leucocytosis of 14,200. The Kahn test was negative.

The urea nitrogen was 13, the blood sugar 91. Examination of the urine revealed nothing abnormal.

When the patient was admitted to the hospital she was seen by another surgeon and it was his opinion that she was suffering from intestinal obstruction. A barium enema, on the day of admission, revealed no evidence of obstruction in the colon, sigmoid or rectum. Two days after her admission a sigmoidoscopic examination was made without revealing any evidence of obstruction, but nevertheless, because of the patient's history and physical findings there was still a strong suspicion that she probably had a malignant obstruction somewhere in the colon. During this time the patient's condition improved somewhat as a result of repeated enemas and the distention almost entirely disappeared. Her temperature, pulse rate and respirations were normal. She still complained of griping abdominal pain.

I saw the patient five days after her admission, at which time examination of the abdomen could be done with a fair degree of satisfaction. The chief feature of this examination was a palpable, very hard round mass below the right costal margin which was slightly movable, not tender and apparently attached to the liver. I suspected that this was probably a metastatic carcinoma of the liver, but in view of the uncertainty of the diagnosis and the clinical evidence of subsiding intestinal obstruction, it seemed advisable that operation should be done.

An upper right rectus incision was made. The gall-bladder was surrounded by dense adhesions which fixed it firmly to the hepatic flexure of the colon. Separation of these adhesions revealed a fistulous communication between the gall-bladder and the transverse colon; the opening in the colon was about the size of a fifty-cent piece. The edges of this opening were trimmed down to healthy tissue and closure made by two rows of catgut suture reinforced by interrupted linen sutures. The markedly thickened walls of the gall-bladder were contracted around one large faceted stone. Because of the inflammatory edema in the region of the cystic and common ducts, the gall-bladder was freed from above downward and then resected, leaving a fairly good sized stump which was oversewed to control bleeding. The gall-bladder bed in the liver was oversewed. A double strip of iodoform gauze was placed against the liver bed, surrounded by three cigarette drains and the omentum interposed between these drains and the transverse colon. The wound was closed in layers around the drains.

The immediate convalescence was uneventful. About ten days after the operation the patient began to complain of severe abdominal cramps which occurred almost every day and on several occasions were associated with nausea and vomiting. These attacks closely resembled intestinal obstruction but they always relieved themselves spontaneously. Because of their severity and tendency to recur I had almost decided to reopen the abdomen. At this time a rectal examination was made and I could feel a hard mass which was movable and seemed to be located in the region of the sigmoid. Another rectal examination two days later revealed a large gallstone in the rectum which was removed after gentle dilatation of the anal sphincter. This was followed by a complete disappearance of the patient's symptoms and within three days she was discharged from the hospital feeling entirely well and with the abdominal wound completely healed. I saw the patient at intervals during the following year. She remained in good health and had no abdominal complaints.

In retrospect, we can reconstruct the events which took place in this patient. She had, to begin with, two large stones in the gall-bladder; the gall-bladder formed an inflammatory attachment to the hepatic flexure of the colon with which it established a fistulous connection; through this fistula one of the stones passed into the hepatic flexure of the colon and because of its size it gave rise to the attack of intestinal obstruction which brought the patient to the hospital. The stone, through repeated attacks of obstruction, finally found its way to the rectum from which it was removed.

Twelve years ago, with the late Dr. E. S. Judd, I reported 153 cases of internal biliary fistula from the Mayo Clinic, the largest single series on record. In preparing this report we had occasion to review the literature and it was astounding to learn the bizarre types of biliary fistula which had

been recorded. For instance, gallstones have been vomited from the stomach; they have been coughed up from the bronchi; they have been voided with the urine and have been found impacted in the male urethra and also removed from the bladder by lithotomy; they have been found in the pregnant uterus, in the contents of an ovarian cyst; they have been found in the pericardial and pleural cavities; and the autopsy performed on Ignatius Loyola revealed that his portal vein contained a number of gallstones. From the review of these cases and a study of the literature the following facts were deduced: (1) The fistula does not cure the disease; (2) it rarely occurs in the absence of stones; (3) the organs involved in their order of frequency are the duodenum, the colon and the stomach; (4) the fistulous connection usually is by direct communication—in other words, rarely is there an intervening abscess cavity; (5) intestinal obstruction is very uncommon in these cases; (6) the diagnosis is made only at the operating table; (7) the mortality is 10 per cent.

Whenever the gall-bladder is adherent to neighboring structures, separation should be carefully done and the freed organs always should be examined for a fistula which may be small and escape detection. It is advisable to remove the gall-bladder whenever conditions permit. The common duct should always be examined for the presence of stones and, of course, when they are found they should be removed.

In recent surgical meetings the subject of the management of acute cholecystitis has come in for considerable discussion. There are those, and they have the backing of the older element of surgical authority, who advocate withholding operative interference until the acute clinical signs have subsided. There are strong arguments, chiefly those of extended experience, to support their opinion. On the other side are the advocates of immediate operation for reasons analogous to factors in acute appendicitis.

I feel that much of this controversy is due to misunderstanding. If an experienced surgeon from each group were to go to the bedside of an individual patient suffering from acute cholecystitis I believe that there would not be much difference of opinion as to the surgical management. It must be understood that the ordinary acute biliary colic does not constitute acute cholecystitis. No surgeon regards this condition as a surgical emergency.

My understanding of acute cholecystitis from the clinical standpoint is as follows: After a period of upper abdominal distress characterized by bloating, impairment of appetite, nausea and vomiting, there is the onset of pain, not very acute, in the right upper abdomen associated with moderate local tenderness and soreness which does not subside in reasonable time. Associated with these symptoms is the general appearance of illness, with a marked febrile reaction, although no chills, and possibly jaundice, after forty-eight or seventy-two hours. The apparent severity of the patient's illness far surpasses the physical findings elicited by abdominal examination.

In a patient of this description I believe that wise surgical judgment is best served by masterful inactivity coupled with close daily observation to detect the sudden appearance of acute complications. If we could look inside the abdomen of this patient what would we see? The gall-bladder would be acutely inflamed, partially distended and surrounded by fresh inflammatory adhesions, bathed in free peritoneal fluid. The liver would be congested and its enveloping peritoneum would be inflamed, particularly in the area adjacent to the gall-bladder. The common duct and cystic duct would be difficult to identify because of inflammatory changes in the surrounding tissue and the glands along the extrahepatic biliary tract would be enlarged. This lymphadenitis and lymphangitis probably would have extended down to and around the head of the pancreas. If the microscopic eye could explore the interior of the liver it would reveal that the acute

inflammation even had extended along the course of the small bile channels within the liver lobules.

Would it be wise in these circumstances to invade this hotbed of unrest in which the forces of nature are waging a successful battle? Is one justified in introducing what from a biologic standpoint is a crude and clumsy procedure, namely, removal or even drainage of the gall-bladder? Can this intervention serve any useful purpose when it is realized that the infection with its inflammatory reaction is not in a subsided or even subsiding stage and when its main focus is by no means localized or confined to the gall-bladder?

One of the arguments advanced for immediate operation in cases of acute cholecystitis is the potential danger of rupture or perforation of the gall-bladder. This is a very uncommon condition and usually when it does occur the surgeon has not had the opportunity to see the patient at the onset of his illness. Rupture of the gall-bladder may, of course take place while the patient is under observation, but when it does it is usually evidence of neglectful observation. Every patient with acute cholecystitis who is under observation should have at least a daily examination of his abdomen with particular attention to the development of acute physical signs. At times the development of disease in the gall-bladder becomes fulminatingly acute in a very short time. This is exemplified in the following report:

One Sunday night a man 60 years of age was referred to my service in the hospital because of acute abdominal pain. He stated that two weeks previously he had an attack of abdominal pain which was not severe and lasted about two hours. On the day before admission he was seized with an attack of severe pain in the right side of the abdomen, followed by vomiting which lasted throughout the day. There was nothing of a pertinent nature in his past medical history and he especially stated that his appetite and digestion had always been of the best.

The chief features of the physical examination were as follows: The abdomen was dis-

tended and peristalsis was active. There was an area of localized exquisite tenderness on the right side of the abdomen somewhat toward the flank and about midway between the costal margin and the umbilicus. This area was very tender to the slightest pressure.

The temperature was 100 and pulse 108. A blood count showed a leucocytosis of 19,250. The urine was normal.

I felt that here was an acute surgical abdomen which demanded immediate operation. Because of the history of rapid onset of acute pain followed by nausea and vomiting and the physical findings, I suspected that we were dealing with acute appendicitis with the appendix occupying a high position.

Operation under spinal anesthesia was carried out a short time after the patient's admission. A mid-right rectus incision was made. Examination of the appendix revealed no gross changes. The omentum was strongly adherent to the parietal peritoneum in the right upper abdomen and could be separated only by sharp dissection. We then saw that we were dealing with a tightly distended thick gall-bladder, the distal part of which was black and gangrenous and in which an area about the size of a fifty-cent piece was of paper thinness, obviously on the verge of impending rupture. The gall-bladder was freed from its surrounding omental adhesions and only then could an examination be made of the region of the cystic duct. This was found markedly thickened by inflammatory and edematous tissue which made it utterly impossible either to identify the important structures in this area or to consider an attempt to remove the gall-bladder. The fundus of the gall-bladder was opened, releasing thick hemorrhagic material and a number of stones of various sizes. One large stone was impacted in the neck of the gall-bladder and was removed only after some difficulty. A split rubber tube of large caliber was placed well down into the gall-bladder and then sutured in place by attaching it with a through and through suture to the opening. The opening was then closed by suture around the tube. Two cigarette drains were placed outside the gall-bladder and the omentum carefully tucked around these. The wound was closed in layers around the drains.

The patient's convalescence was without incident. The tube in the gall-bladder came away spontaneously on the tenth day and the cigarette drains had been removed three days

previously. Bile drained freely from the tube and later from the wound and continued to do so for about twenty days. The patient was discharged from the hospital twenty-five days after the operation with his wound soundly healed and apparently in good general condition. I saw him at my office at intervals for eight months after his discharge from the hospital during which time he remained entirely well.

A married woman, aged 48 years, was admitted to the hospital February 1, 1938 because of attacks of biliary colic associated with jaundice. She had her first attack twelve years before, beginning as a crunching pain in the epigastrium and radiating around the costal margin to the angle of the scapula. It was followed by jaundice which lasted four or five days. The next attack occurred nine years later and was similar to the first attack. During the past three years she had been experiencing attacks of severe biliary colic always followed by jaundice and occurring about every three or four months. The last episode occurred five months before admission and for the first time it was associated with chills and fever and jaundice of one week's duration. In twelve years the patient's condition between attacks had been characterized almost continuously by heartburn and epigastric distress, consisting of fullness and bloating especially after eating greasy and fried foods.

There was nothing of a relevant nature in her past medical history. A cholecystographic study made a month before admission showed the presence of gallstones. The physical examination revealed nothing of importance except tenderness on deep pressure directly over the gall-bladder. The temperature, pulse and respiratory rate were normal. There was no evidence of jaundice. The examination of the urine was normal. The blood count showed a hemoglobin of 13 mg., red blood cells 4,100,000 and white blood cells 14,000.

Operation was carried out ten days after admission under ether anesthesia. An upper right rectus incision was made. The liver presented a nutmeg appearance and the edges were rounded. The gall-bladder was hugely distended and contained many stones. The common duct was dilated to a diameter of 5 cm. and was tightly packed throughout by stones and sand and these extended also up into the main hepatic duct. The common duct was opened through a longitudinal incision and at once thick dark bile with stones of various sizes and sand gushed forth. The common duct and the hepatic duct were emptied of their contents with the aid of scoops and forceps and finally by flushing with normal saline through a rubber catheter. Both ducts could be easily explored with the finger throughout the entire extent so that one could feel fairly sure that no more stones were left behind. The head of the pancreas was not enlarged. A No. 20 French catheter was placed in the common duct with its end pointed upwards in the hepatic duct and fastened in place by a suture of catgut. The opening in the duct was then closed around this catheter and bile began to drain freely from the tube.

The gall-bladder was removed from below upward and the gall-bladder bed oversewed with catgut. The stomach and duodenum were normal. The appendix was atrophic and was not disturbed. The uterus contained a number of small fibroids. A cigarette drain was placed in the subhepatic fossa below but not in contact with the common duct. The wound was closed in layers around the two drains.

The patient's postoperative convalescence was uneventful. At first the bile draining from the catheter was a dark muddy brown with a heavy sediment but later it became a light syrupy golden yellow. The amount of bile drainage varied between 450 and 650 c.c. daily. The color of the stools beginning with the first bowel movement on the fourth day was a normal brown color. The cigarette drain was removed on the eighth day and the tube in the common duct came away spontaneously on the fifteenth day. The patient had two attacks of auricular fibrillation which yielded promptly to digitalis therapy. The patient was discharged on the twenty-third day with the wound completely healed. At the last examination two months after the operation she was entirely well.

The noteworthy features in this report are: (1) the enlarged distended gall-bladder containing stones associated with numerous stones in the common duct; (2) the absence of jaundice for five months preceding operation in spite of the fact that the hepatic and common ducts were tightly packed with stones and sand; (3) the huge distention of the common duct which measured 5 cm. in diameter.

The association of a contracted gall-bladder with stone in the common duct is a surgical maxim that has come down through the years. The necessary time for the development of jaundice in the event of obstruction of the common duct is considerably shorter when the gall-bladder is functionless through disease or absent than when it is present and capable of receiving and concentrating bile. Nearly all stones in the common duct have had their origin in a diseased gall-bladder and this accounts for the advanced cholecystic changes. However, there is ample presumptive evidence of the formation of stones of successive vintages in the common duct itself. Perhaps the first one or two stones formed in the gall-bladder, passed through the cystic duct into the common duct where their presence gave rise to partial or intermittent obstruction with inflammatory changes. So far as we know this combination of circumstances is most conducive to the formation of gallstones.

I believe this to be the explanation of the great number and varied sizes of stones in the common duct of this patient. The hepatic and common ducts were hugely dilated and literally packed with stones which varied in size from about 2 cm. to fine sand. Yet in spite of these many impediments to free passage, the bile succeeded in percolating through this mixture of gravel in sufficient amount to prevent the development of jaundice except after attacks at long intervals.

SUMMARY

Advanced disease of the biliary tract gives rise to surgical problems in which disturbed physiology, mechanical factors and infection play interdependent rôles. Four surgical cases are reported and discussed:

1. Obstructive jaundice caused by stone in the common duct.

2. Internal biliary fistula.

3. Acute gangrenous cholecystitis with stones.

4. Calculous common duct without jaundice.

PROGRESSIVE POSTOPERATIVE GANGRENE OF SKIN*

REPORT OF A CASE

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IN the course of postoperative complications we have observed a hitherto rare disease—a slowly progressive gangrene of the abdominal wall. According to reports in the literature, Cullen was the first to observe the condition, after the removal of a suppurated appendix, in 1924. The same year Christopher reported a similar process following rib resection for empyema thoracis. In 1926, Brewer and Meleney, Mayeda, Diebold, and Stewart-Wallace dealt with the question. Küppers, Doricev, Coacley and Klein, Branberg, Andersen, Liedberg, Cox, Blaxland, Willard, Wachs and Guszman contributed to the clearing up of this disease with case reports and, in 1935, according to Stewart-Wallace, thirty-seven cases had been reported.

At this writing, the number of reported cases is about sixty, (1924–1939) which is rather low. Probably some of the fatal cases have not been published.

As new evidence shows that we are dealing with a clinical entity, it would be advisable to keep the nomenclature uniform in order that all cases may be followed up. Brewer and Meleney were the first to call the entity "progressive postoperative gangrene of the skin," which seems suitable as it expresses the essence of the process.

This lesion usually occurs in the male (about 80 per cent), especially in the adult. The preceding operations were, in one-half of the reported cases, due to suppurated appendices, in 30 per cent to empyema thoracis, in 15 per cent to suppuration of the upper abdominal cavity, gastric perforation and subphrenic abscess and in the remaining 5 per cent to other operations, mainly abdominal. It was reported in but three instances after aseptic abdominal operations.

The process usually begins within a week following the operation, and occasionally during the second week. One observation was made twenty-one days after operation. Often the fever was gone, the pulse normal and the patient well; in the case reported by Patterson, the patient had been discharged from the hospital.

CASE REPORT

A male, aged 45 years, was admitted to the clinic on October 28, 1937. Months before, the patient had complained of severe pains in the abdomen and for one month of loss of weight due to polyarthritis. Two days before admission he had severe abdominal pain which soon localized in the lower right side of the abdomen. He suffered from nausea, vomiting, fever and chills, and had lost much weight.

There was marked muscular rigidity of the abdomen especially around McBurney's point and there was an apple-sized, hard, painful resistance at this point. The Rovsing symptom was positive. The white blood cell count was 17,000, the temperature 38°c., the pulse 110. The tongue was coated and furred. Immediate operation was deemed necessary.

A right McBurney incision was made. Behind the cecum a fetid abscess, the size of a fist, was opened and drained with gauze. The abdominal wall was closed with interrupted sutures about the gauze drains.

For a week following operation, the patient was very ill, hiccoughing and vomiting. His abdomen was greatly distended, but enemas were ineffectual. The wound showed a profuse drainage of pus. At the end of the week the hiccoughing stopped and gas was passed. Nineteen days after operation the wound showed an area of edema, the edges became separated and were reddened and painful. The redness spread toward the healthy parts of

the skin and necrosis was observed in the wound edges. Twenty-three days after operation, the subcutaneous connective tissue of the wound cautery was used on January 8 and four days later a blood transfusion was given. From January 13 a protracted hypermanganous bath

FIG. 1.

was granulating well, but the necrosis of the skin margins already extended 4 mm. from the edges of the wound.

On November 26, the wound was very painful and necrosis of the skin had spread to a distance of 6 mm. As this zone became demarcated, part of the necrosis was removed by scissors. The gangrene of the wound continued to spread and on December 7, the wound was treated with 100 Roentgen units. A culture taken from the wound on December 14 showed streptococcus and proteus. Blood examination revealed: Red blood cells 3,900,000 and white cells 11,300, hemoglobin 77 per cent; polys 76 per cent, lymphocytes 17 per cent, eosinophiles 2 per cent, basophiles 1 per cent and mononuclears 4 per cent; 2 plasma cells per 100 white blood cells.

Operation was performed on December 22. The gangrenous zone spreading from the edges was entirely removed by the Paquelin cautery.

Histologic report on the specimen, 12 cm. of necrotic tissue, showed gangrenous fibrous tissues infiltrated with leucocytes, in a state of chronic inflamed infiltration, with purulent ducts and many microbes present.

Aerobic cultures showed proteus; anaerobic cultures showed *Streptococcus putrides.*

The purulent discharge continued and wet zephyrolous dressings were therefore used daily. On December 28, six tablets of sulfanilamide were given. The wound progressed satisfactorily until January 3, when new gangrenous foci at three different points on the medial aspect developed. The electro-

followed by a zephyrolous dressing was given daily. Gangrene, however, spread in all directions. (Fig. 1.)

After a consultation by Professors Ádám, Zalka, and Guszman, operation was performed. Far into the intact zone to the muscles, the diathermic knife was carried around the whole necrotic area. Zephyrolous dressings were used daily, with the hypermanganous bath every second day. Twenty units of insulin were given. On February 7 a new focus on the medial aspect was burned out, and five days later a new focus on the lateral side was cauterized into the healthy zone. By February 18 extension had stopped, the wound edges cleared, the bottom of the wound was red, filled with clean granulation, and here and there islands of epidermis appeared. (Fig. 2.)

Thereafter the patient made a speedy recovery and on March 16, apart from a few small areas which had to granulate, the wound was epithelized. The patient was then discharged. The wound was completely healed May 5. (Fig. 3.)

Pathology. The necrosis of the wound edges begins slowly, at the beginning involving only small areas—sometimes in areas of deep sutures. An ulcer involves the skin edges which are red, edematous and painful. Soon the necrosis spreads over the connective tissue and deeper layers. The wound edges become elevated, thickened,

and serpiginous in outline, owing to the necrotic process of the subcutaneous tissue, the skin giving a carbuncle-like

back from the neck to the buttocks became ulcerated.

Histologically, according to Mayeda, the

FIG. 2.

FIG. 3.

picture. The extensive infiltration thereupon changes over to dry type of gangrene. After the removal of the gangrenous zone, a relatively healthy red granulating tissue remains in its place. While the ulcerous process continues, serpiginous healing is to be seen on other areas, sometimes on the isles. The extension occurs slowly, maliciously and usually spreads. Cox distinguishes four zones of extension: (1) outside, painful infiltration; (2) 0.5 cm. sharp demarcated margin of black gangrene toward ulcer; (3) ulcer-ground dirty, grayish-white; (4) well granulating healthy red tissue. The greatest extension observed was in Poate's patient, where the whole

necrosis destroys first the border between the papillary and the reticular stratum of the skin. Part of the epidermis and reticular stratum covers the gangrenous mass to a depth of 1.5 cm. and slowly necrotises from the wound edge. A part of the reticular stratum remains intact and in the subcutaneous fat tissue there is some leucocytic infiltration. Where gangrene ends and granulation begins there are a few islands of epidermis arising from remainders of sweat glands which provide a partial basis for later epithelization. According to Wachs' serial histologic examinations, the process is inflammatory and soon leads to necrosis. The infiltration consists mostly of

lymphocytes and greatly surpasses the necrotic border. The skin and papillae quickly fuse, and coagulated fibrin and

gangrene of the skin developed on the buttock at the site of injection and spread over the thigh. They believe that the

FIG. 4.

lymphocytic infiltration cover the ulcerated area. Nerves and arteries remain intact; the extensive infiltration around the veins is remarkable, even in parts far beyond the inflammatory focus. Many giant cells are present. Thrombophlebitis has been found, not only in the ulcerated zone, but also in the intact area, involving the intima early and later the other coats of the vessel as well. The border is not sharp. Many staphylococci, streptococci and Gram-negative bacilli are to be found in the ulcerated zone.

Etiology. In most cases streptococci have been found, and, rarely, staphylococci, diphtheroids or Gram-negative bacilli. Heimburger, Cole and Heideman described cases where the principal role was attributed to the ameba, but this has not been noted by other authors. Most of the patients were debilitated, but no antecedent specific disease, such as lues or diabetes is reported. Lynn attached great importance to the icebag on the abdominal wall as a causative factor, while Borelli attributed the condition to tincture iodine and Tennant to liver insufficiency. In the case of Gatewood and Baldridge, prophylactic scarlet fever serum was given and scarlatina developed. Great doses of serum were then administered and in consequence

second injection led to gangrene in a hypersensitive organism (Arthus phenomenon). Ballin and Moore supported this view by experiments on animals. However, these various theories have received no confirmation.

The most acceptable etiologic theory is that of Meleney who, after experimentation, explains the development of the gangrene by bacterial synergism, with the streptococcus chiefly involved. Meleney isolated the so-called micro-aerophyl nonhemolytic streptococcus (*Streptococcus evolutus Prévot*), from the extending zone. In the gangrenous zone the streptococcus was associated with *Staphylococcus hemolyticus* and diphtheria bacillus. The diphtheroid bacillus was not pathogenic for animals; nor was the streptococcus or staphylococcus alone, but, when injected together, they caused a process similar to that in humans. Apparently in the extending zone the ground is prepared by the streptococcus, but for the development of the process, symbiosis with other microbes is necessary.

In our case the facultative anaerobic streptococcus with proteus in symbiosis was found. In the cases of Mayeda, Cole and Heideman, Stewart-Wallace and others, necrosis began in deep suture holes where

anaerobic conditions exist. Patterson now omits, for this reason, the use of deep sutures. In our case, however, such sutures were not used.

According to Wachs, thrombophlebitis is the basis of the gangrene and spreads into the intact area. No other author has reported this. In the present case (Fig. 4) the veins showed no change beyond perivascular infiltration. In one of Wachs' cases a varicose ulcer had existed for two years; its sudden spread necessitated amputation of the leg. It is quite possible that thrombophlebitis appeared in the varicose veins and spread, causing a considerable progress of the ulcer. However, it is less probable that this disease had any connection with the skin gangrene. In Wachs' other case, a severe inflammation began in a small infection on the hand. Gangrene spread despite the disarticulation of the fourth and fifth fingers and led to the destruction of the back of the hand as well as a great part of the hypothenar. This case does not appear to have been caused by thrombophlebitis.

It is our belief that the gangrene is due to the direct lytic effect of the microbes.

Symptoms. The clinical symptoms correspond to the previously described pathologic-anatomic process. The most characteristic symptom of gangrene of the skin is severe pain, most marked on the edges of the wound. The temperature is usually subnormal, although, with the spread of the lesion higher temperatures may appear. Anemia, exhaustion and mental depression develop with the extension of the gangrene. The process is not contagious.

Diagnosis. Diseases which show some similarity to postoperative gangrene, according to Stewart-Wallace, are:

1. Wound infection, where the suppurating infiltration may pass over into necrosis if the cause of infection is especially virulent and the resistance of the patient is low. This might extend to the deeper layer of the abdominal wall, and is the so-called "hospital gangrene" which was not rare before the aseptic period. Skin gangrene differs in that it destroys only the subcutaneous tissue and the skin, not extending to the deeper layer. It is not contagious.

2. Erysipelas in its early stages may be similar, but does not cause a spreading ulcer.

3. Gas gangrene is followed by more serious general symptoms with subcutaneous crepitation and with characteristic bacteriologic findings.

4. Hemolytic streptococcal gangrene of the skin (Meleney) is observed mostly in China, but recently has been seen in New York. This is usually observed on the thigh, sometimes on several places at the same time, with characteristic blood culture and symptoms of exhaustion.

5. Ecthyma gangrenosum is particularly observed in undernourished children and is often multiplex.

6. In specific infarctions, diphtheria, blastomycosis, tuberculosis, etc., the demonstration of the cause of the disease simplifies the differential diagnosis.

7. Amebiasis cutis has been reported only by Heimburger, Cole and Heideman. They believe that a good reaction to an injection of emetin hydrochloride is characteristic, as are the bacteriologic findings.

8. Gangrene may start following human bites, if the deeper tissues are infected by saliva. The presence of fusiform bacillus and spirochetes is characteristic.

Treatment. The general opinion is that the only treatment is surgical. Only four cases have been reported as cured without operation. One of these healed in consequence of quartz light (Gordon); one by treatment with 6 per cent sodium chloride (Clinton); one after immunized blood transfusion (Probstein and Selig); and one by the use of maggots (Coakley and Klein). Experiments were made with the local use of various antiseptic and hypertonic solutions, with vitamin diets and creams, with the injection of arsenic, calcium, manganese, antimony, salvarsan, and of specific and nonspecific serums, heteroprotein vac-

cine, with blood transfusions and Roentgen therapy—all without results. The only sure method is the excision of the gangrenous wound edges far into the intact zone, producing healthy wound surfaces. Some authors recommend a knife and some the electrocautery. For styptic action and for cutting off lymph passages, we consider the electrocautery the better.

Little may now be expected from non-surgical treatment, but we wish to call attention to the use of sulfanilamide which we believe was beneficial in our case. We found also that the use of zephyrolous dressings reduced the quantity of secretion greatly and stimulated the formation of healthier granulating tissue. Blood transfusion may be useful for the recovery of the exhausted patient.

In many cases Thiersch grafts have been used to supply the missing skin. In our patient this was not necessary as the deficiency was soon supplied, partly from the edges and partly from the isles of epidermis in the affected region. It is most interesting that the surface of the wound becomes much smaller and that there is but little contraction of the scar. The cause lies in the sweat glands which remained intact and which supplied some well adhered pinch grafts capable of development.

We consider the 5 per cent mortality of all the collected cases by Lynn to be too low, but the prognosis cannot be called bad if early recognition and suitable treatment are possible. As to prophylaxis, the sugges-tion of Meleney that deep sutures should not be used on suppurated abdominal and thoracic incisions, we do not deem necessary in view of the rarity of the complication.

SUMMARY

A case of progressive postoperative gangrene of the abdominal wall has been presented. Hitherto some sixty cases have been reported.

The process is a well outlined entity showing itself in the progressive necrosis of the skin and subcutaneous connective tissues, developing chiefly after operations on suppurating abdominal and thoracic lesions, and produced by symbiosis of specific streptococcus and nonspecific bacteria.

Severe pain is characteristic. The gangrene does not extend to deeper layers and it is not contagious.

Surgery is the only effective treatment. Postoperative treatment consists of sulfanilamide, zephyrolous dressings and blood transfusion. Early diagnosis is important and makes for a better prognosis.

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ACUTE NECROSIS OF THE KIDNEYS

REPORT OF A CASE

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BILATERAL cortical necrosis of the kidneys is rare. One of the earliest cases was reported in 1886 by Juhel-Renoy[1] of France. The patient was a girl 16 years old who had scarlet fever and died eight days after the onset of symptoms. Evans and Gilbert[2] added a case to the literature in 1936 and stated in their article that about fifty-three proved cases had been recorded, for the most part by German and English authors. Only fourteen cases have appeared in American medical journals. Evans excluded all cases in which a post-mortem examination was lacking, or when biopsy was done on one kidney only.

In these cases of acute necrosis of the kidneys the etiologic factors were pregnancy (77 per cent), malaria, grippe, scarlet fever, diphtheria, acute perforated appendicitis with peritonitis, acute poisoning, hog cholera, cerebrospinal fever, acute polyarteritis nodosa, pulmonary tuberculosis, pneumonia, carcinoma of the prostate gland, and intravenous camphor.

In most of the cases reviewed there was complete suppression of urine of from two to ten days' duration and death was due to acute renal failure. The uremic phenomena occurred late, usually about the seventh or eighth day of the anuria. Von Zalka,[3] in a review of thirty-one cases in pregnant women, found a uniform clinical picture of eclampsia—convulsions, vomiting, edema, and coma. The urinary output diminished rapidly, with a corresponding rise in the non-protein nitrogen content of the blood. However, in Davidson and Turner's[4] report of four cases in pregnant women there were no convulsions, no suggestion of arteriosclerosis, and no signs of chronic interstitial nephritis. In one case only, post-mortem examination revealed minute areas of degeneration in the liver in addition to cortical necrosis of both kidneys. There were no signs of previous renal damage. In three instances the spleen was congested. Hemolytic streptococci were cultured from the spleen.

In discussing these cases, Morison[5] stated that he had seen this condition only once, when he observed acute necrosis of the cortex of the kidneys in an experimental animal. Saunders and Hartman[6] conducted 100 autopsies on patients who had died of uremia. Several of the kidneys examined showed slight scarring but none presented evidence of acute necrosis.

Because of its rarity and in view of the fact that a search of the literature failed to disclose a case associated with acute pancreatitis, the following case of acute cortical necrosis of the kidneys is reported.

R. W., a white male age 38, was admitted to the hospital October 1, 1937. His illness began five days before entry, on September 26, while he was at the American Legion Convention in New York City. At this time he had what he described as an attack of acute indigestion. He complained of severe upper abdominal pain associated with belching of gas, frequent attacks of vomiting, and diarrhea. On the first day of his illness he noticed blood clots in the urine. The following day he did not void, and no urine was obtained by catheterization. From the onset of the attack he was unable to retain food. Abdominal pain and diarrhea persisted. Therefore he was referred to the hospital.

This patient had had epilepsy since 1918, with some attacks of the grand mal type. Otherwise his past history was non-contributory.

Physical examination revealed a well-developed and well-nourished, but highly nervous man, with marked tremor of the hands. His temperature was 101.8 degrees, his pulse rate 82, respirations 36. His blood pressure was 110/75. There was moderate tenderness in the

epigastrium and over the urinary bladder. The abdomen was distended. There was no edema. Heart and lungs were normal. The

fluid was withdrawn. The left ureteral orifice was obscured by blood clot and fibrin.

After this examination the abdominal dis-
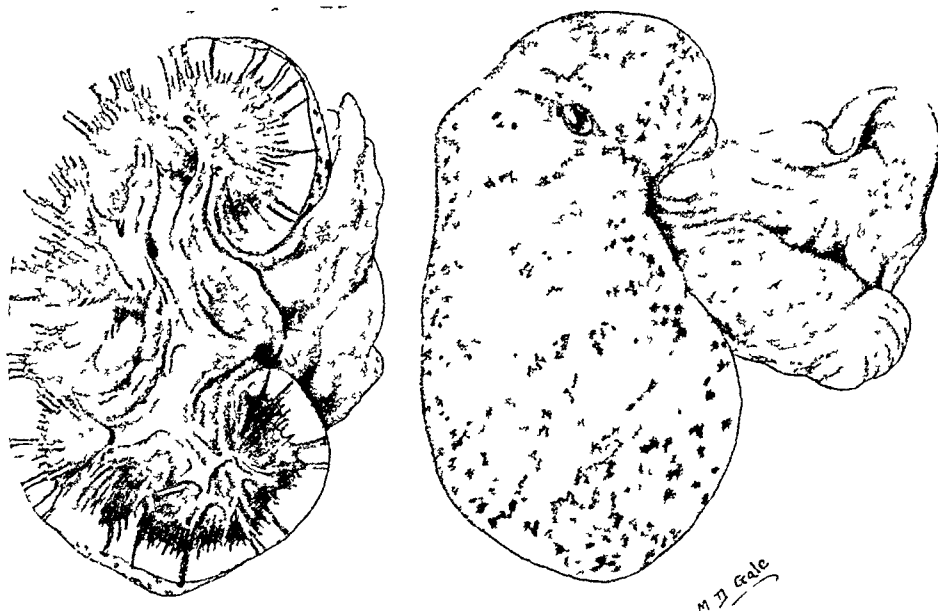
FIG. 1. Drawing of kidney showing gross appearance. Cross-section shows appearance of cortex and medulla. Dark areas indicate the only remaining viable tissue.

white count was 9,900. The red cells showed very slight anisocytosis. The non-protein nitrogen was 171 mg., the creatinine 7.5 mg.

The following day, October 2, a more complete blood examination revealed 3,850,000 erythrocytes, 8,500 leucocytes, and 69 per cent hemoglobin. The differential count showed 80 per cent neutrophiles, 15 per cent lymphocytes, 4 per cent large mononuclears, and 1 per cent eosinophiles. There was very slight anisocytosis.

The blood chemistry was as follows: blood sugar 142 mg., non-protein nitrogen 181 mg., creatinine 8.5 mg.

A flat plate of the renal tract made October 1 showed the kidneys to be normal in size, shape, and position. There was no evidence of renal calculus. A barium enema the following day showed no evidence of large bowel pathology.

On October 2 a cystoscopic examination was performed under spinal anesthesia. A Brown-Berger cystoscope was easily passed into the bladder. The trigone was injected. The trabeculations of the bladder mucosa were prominent. No stones or tumors were seen. A No. 5 catheter was passed up the right ureter for a distance of 25 cm. without meeting any obstruction. A small amount of sanguinous

tention decreased to some extent. Two hours later he voided an indeterminable amount of what was considered residual fluid following the irrigation of the bladder. His general condition did not improve. He was treated daily with 3,000 c.c. of 10 per cent glucose administered intravenously and was put in an oxygen tent. His temperature remained between 102.4 and 103 degrees and his pulse rate between 100 and 120.

The blood chemistry determinations were as follows:

Date	Non-protein Nitrogen, Mg.	Creatinine, Mg.
October 2	181	8 6
3	188	8 7
4	194 5	8 6
5	200	8 8
6	207	12 0
7	222	12 0
8	240	12 5

The patient gradually became more stuporous and respirations more labored. He finally lapsed into a uremic coma and died on the seventh hospital day, due to complete anuria secondary to an acute nephritis, etiology unknown.

Post-mortem examination revealed an acute pancreatitis with extensive areas of fat necrosis throughout the abdomen and an almost com-

plete necrosis of the cortex of both kidneys. No other etiologic agent was discovered for the anuria except the pancreatitis and its resultant

injury to the kidneys was secondary. The pathology of the kidneys was that of infarction, the injurious agent affecting

FIG. 2. Section through junction of cortex and medulla showing darker-staining necrotic area and suggestive thrombus in a small vessel adjacent to a glomerulus.

FIG. 3. Section through the subcapsular portion of the cortex showing a small island of tubules which have not been destroyed, probably obtaining their blood supply from the capsule.

toxemia. The appearance of the kidneys is shown in Figure 1. The right kidney weighed 200 Gm., the left 180 Gm. The cortex measured 5 to 6 mm. The surface of the kidneys showed a number of reddish areas, which represented the only viable tissue in the cortex. The medulla of both kidneys was congested.

Microscopically, the entire cortex showed necrosis of the coagulative or infarct type. At the junction of cortex and medulla there was a polymorphonuclear cell infiltration in the necrotic portion. (Fig. 2.) The involved areas had undergone such a marked change that it was impossible to locate the smaller thrombosed arteries definitely. However, near the junction of the cortex and medulla several larger interlobular arteries were noted. These were filled with necrotic hyaline-like thrombi and the walls of the vessels were also necrotic. Some of the glomeruli at their edges showed only partial or beginning necrosis amid the more normal tubules. Thrombosis of the afferent arterioles was not seen in these early lesions. Beneath the capsule there was a small margin of normal tissue which had probably obtained its blood supply from the capsule. (Fig. 3.)

Thus, the microscopic study revealed acute necrosis of both kidneys and pancreatitis of moderate duration. The fibrous tissue proliferation about the areas of fat necrosis indicated that the first symptoms were due to pancreatitis and that the

the arterioles of the cortex. Acute necrosis of the kidneys is secondary to various toxic states, and in this instance the etiologic factor was a pancreatitis of less than usual severity. The blood sugar level was an indication that the pancreas itself suffered only moderate loss of function. No cause of the pancreatitis was elicited. The gallbladder ducts and the pancreatic ducts were patent throughout.

The microscopic descriptions of cortical necrosis which have appeared in the literature correspond with the findings in this case. Many of the glomeruli and afferent and efferent vessels contained fibrinous thrombi. The convoluted tubules contained casts and red blood corpuscles. No endarteritis was seen in the renal arteries and the medulla showed no necrosis.

The question as to where the toxic injury attacks the endothelium is debatable. The glomeruli may be injured first and thrombosis occur in the interlobular arteries; or the arteries may become blocked first with secondary injury to the glomeruli. Jardine and Kennedy[6] in one case found thrombosis only in the capillaries and smaller arteries after two days of anuria. Therefore they concluded that thrombosis began in the

interlobular arteries, not in the glomeruli. Von Zalka, Glynn and Briggs[7] also agreed that thrombosis began in the interlobular arteries, the walls of which became necrotic and showed fibrinous changes. According to their theory, the thrombi moved distally and finally reached the hila of the glomeruli, thus producing necrosis of the cortex. The objection has been raised that if thrombosis began in vessels as large as the interlobular arteries, some changes ought to occur in the spleen. Further study is needed before any conclusions as to the origin of the thrombosis can be reached.

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A PATIENT should be kept under observation for three days after the application of a cast; if swelling occurs underneath the cast, constriction of the circulation resulting in gangrene or contractures may occur within a few hours.

From—"Fractures" by Paul B. Magnuson, 3rd Edition (Lippincott).

CARCINOMATOUS MASTITIS

CASE REPORT

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CARCINOMATOUS mastitis is an atypical form of breast cancer which, although very malignant, presents some of the aspects of acute inflammation often so closely as to make differentiation from an inflammatory process quite difficult. It has been alluded to as inflamed cancer, acute brawny cancer, carcinoma mastitoides and mastitis carcinosa. The condition is rapidly progressive and is often improperly treated by fomentations and injudicious incisions on account of the accompanying and confusing inflammatory phenomena. Because of its comparative rarity (1.3 per cent of all breast cancers), this case report is submitted.

Bell, in 1841, first drew attention to this atypical condition. Klotz, Volkmann and Billroth later described it. Schumann, in 1911, reported eleven cases and gave it the name carcinoma mastitoides. He observed that most cases, but not all, arose during late pregnancy or lactation. Lee and Tannenbaum, in 1924, reported twenty-eight cases in detail. They emphasized its distinction from other types of breast cancer and held that irradiation is of value in treatment, being palliative and definitely prolonging life, and is to be preferred to surgery. Fox, in 1930, also stressed the value of irradiation instead of surgery in attacking this condition. Orbach, in 1931, recommended irradiation postoperatively. Chevalier, in 1932, reported a case of acute bilateral carcinomatous mastitis. The extensive involvement precluded any treatment. Hartmann, Bertrand, Fontaine and Guerin (1935) reported ten cases. These were irradiated postoperatively, but the ultimate prognosis was invariably bad. Rubens and Duval in the same year reported six cases. They held that protein therapy combined with irradiation was more efficacious than surgery.

CASE REPORT

Mrs. G. H., age 33, was delivered of a full term infant on October 21, 1936. About the sixth puerperal day she developed a "lump" in her right breast which was diagnosed "blind boil" by her attending physician who discharged her on the fourteenth day postpartum. The lump subsequently became more painful and was incised under ethyl chloride anesthesia (spray) four weeks later. Some pus was evacuated. Further incisions were made during the following five months but the lesion never healed. The drainage continued.

The patient consulted me on April 7, 1937. The right breast was moderately enlarged, red and hot. The skin was indurated. A large rosette of granulations was present below and to the right of the nipple and a large sinus draining foul-smelling pus penetrated this rosette. Axillary glands were not palpable. A diagnosis of acute suppurative mastitis was made, and under evipal soluble anesthesia intravenously the area was widely incised and drained. Necrotic breast tissue and some foul-smelling pus were evacuated. No histologic study was made at this time.

The patient was discharged in one week, but readmitted on April 24, 1937 for more extensive drainage. Histologic study of the necrotic breast tissue removed was contemplated but the material was inadvertently lost. The patient was again discharged on May 2, 1937. The condition progressed rapidly, the lesion increasing in size and the discharge becoming more profuse. The right axilla presented enlarged, tender, stony hard glands. Mastectomy (Stewart incision) was done on May 12, 1937. The axilla was not explored on account of the extensive infection. May 29, 1937, the incision was practically healed and the patient left the hospital.

Pathologic examination revealed an adenocarcinoma of the breast, grade iv. There was

an open ulcerating area beneath the nipple and several other openings containing a drain. On gross incision there was an open cavity surrounded by firm neoplastic tissue which

clavicular and right axillary regions, and over the posterior mediastinum. Generalized metastasis and cachexia supervened gradually. Death occurred in April, 1938.
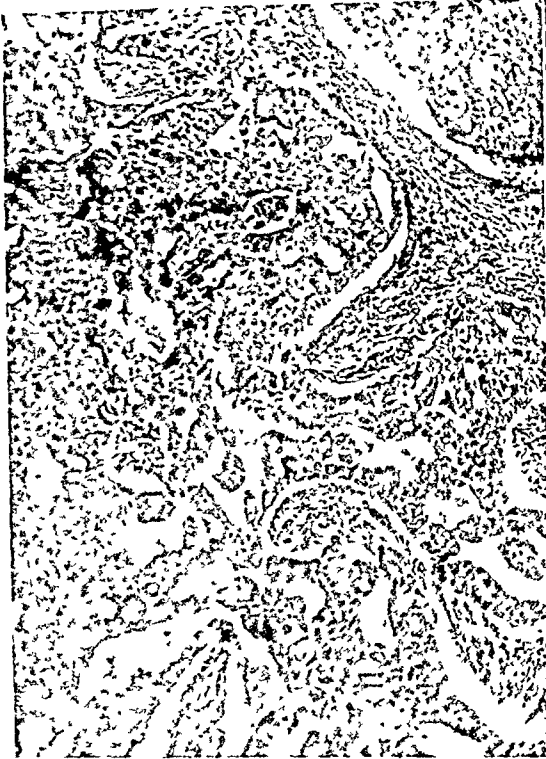
FIG. 1. Low power photomicrograph of carcinomatous mastitis.

FIG. 2. High power photomicrograph.

was granular. Section of the tumor mass showed a thick wall of rapid growing epithelium about the abscess cavity. Many mitotic figures were noted. Acute exudate was found in the stroma and forming the surface of the abscess cavity. Several sections from other parts of the breast showed hyperplasia of the mammary gland with chronic inflammatory infiltration.

Deep x-ray treatment with a total of 5600 R was given over the right breast, right supra-

The marked inflammatory phenomena present in this patient were striking. It was felt at first that the very small incision made under ethyl chloride spray anesthesia was responsible for inadequate drainage and therefore for prolonging the inflammatory process. Only after more extensive drainage was instituted with resultant exaggeration of the lesion were our suspicions of malignancy aroused.

TRAUMATIC DIABETES

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THE question of the causal relationship of trauma to diabetes mellitus has received considerable attention, and is still an open one. Opinions in the various texts on the subject are often contradictory, and one finds authors denying the possibility on the one hand, yet citing instances in which they are inclined to accept trauma as the cause for the diabetes. It involves the larger problem of neurogenic diabetes, which dates back to Claude Bernard's classic experiment, of producing glycosuria and hyperglycaemia in animals by puncturing the floor of the fourth ventricle. This experiment has never been confirmed in man, but we know that organic disease of the brain such as tumors of the pons, medulla and cerebellum, may produce true diabetes. Dresel and Lewy point to diabetes in cases of organic brain diseases, as tumors or hemorrhages either in the sugar center or near it, or cellular changes in the sugar center.

Joslin dismisses the question briefly, admitting that trauma may aggravate diabetes, but denying a causal relationship. He would discard reports in the older literature because of insufficient proof of previous non-existence of diabetes. He quotes von Norden's very decisive statement based on the experiences of the World War which, he says, once for all bury the question. Yet, in a later discourse, he is not quite so emphatic. Grafe concludes rightly, we feel, that the question must be left to clinical and statistical experiences and that "in a few selected cases the connection between neurogenous and traumatic influences and diabetes cannot be denied."

It is apparent that from a medicolegal standpoint this constitutes an extremely important problem, and it is true that it comes up for debate every so often in litigations concerned with liability and double indemnity insurance. The German courts recognize neurogenous diabetes if the following facts are demonstrable: (1) that there are no facts in the case which would indicate the presence of diabetes before the accident; (2) that the interval between the trauma and the onset be short; (3) that the trauma, especially if mental, be severe.

In a large clinic such as ours one encounters many cases in which the etiology, and particularly the mode of onset, appears obscure, and now and again one obtains a history of trauma, which seems to be definitely responsible for or, at least, a contributing factor in the appearance of the diabetes. Ebstein obtained a history of external injury in six of 116 diabetics, and Williamson in six of 100. In more than half of the cases the head was the site of the injury. There is no proof in any of the cases, however, that diabetes did not exist before the accident. How difficult it is to evaluate such facts is shown by the statements of two authors. Gissel observed hyperglycemia up to 207 mg. for three days following head injuries resulting in coma. In a summary in 1930, R. Stern states that a severe head injury has rarely been followed by a true diabetes.

The pathologic changes described in head trauma are significant, and the onset of diabetes in these cases might be explained by them. Gotten, in a recent review of the subject of head trauma, quotes Winkelman and Eckel, who demonstrated hemorrhages in the brain with subsequent edema, and destruction of brain tissue

around the areas of the petechial hemorrhages. Cassasa described multiple petechial hemorrhages throughout the cortex in patients who had no lacerations of the scalp, nor any fractured skull or contusion of the cortex. Gotten concludes that there is "sufficient evidence that even in cases of mild head trauma hemorrhage occurs, with resultant areas of softening and gliosis of the entire brain." In view of the findings of Dresel and Lewy, mentioned above, a pathologic basis for traumatic diabetes might be established.

We wish to report two cases of diabetes in which trauma played a part.

Case i. D. M., a white female, age 26, was admitted to the surgical service on March 4, 1936. She had been struck by a street car and thrown to the ground, remaining unconscious for forty-five minutes. She was married, had two children, seven and three years old respectively; there had been no other pregnancies. She had always been obese, and for several months prior to her admission had received injections of antuitrin-s. Her physician informed us that repeated urinalyses proved negative for sugar and that the last analysis was made one week prior to the accident. In November, 1935 (about four months before the accident) she passed a life insurance examination which included urinalysis. Her maternal grandmother had had diabetes.

On admission she was coming out of her stupor, but did not recall the accident. Temperature was 100.6, pulse 110, respiration 26, blood pressure 124/80. There were three extensive lacerations and contusions of the left side of the face. The pupils were equal and reacted normally; no blood or spinal fluid was noted in the aural orifices. Urinalysis was negative. A spinal tap yielded 15 c.c. of frank blood, under normal pressure.

She was given 1,500 units of tetanus antitoxin and 50 c.c. of 50 per cent glucose intravenously. X-ray examination of skull and chest proved negative. Neurologic examination showed no extraocular palsies, fields and fundi normal, cranial nerves normal. The diagnosis of cerebral concussion was made.

Two days after admission, seventeen sutures were removed from the scalp, and the wounds were found clean. Urinalysis on March 6 was negative. On March 8, four days after the accident, the patient complained of excessive thirst and frequency of urination. A fasting blood sugar taken that day was 278 mg. This report was overlooked by the surgeons, and the patient was discharged on March 18, with a diagnosis of cerebral concussion and multiple lacerations of the scalp.

After leaving the hospital she noted further polydipsia, polyuria, and a loss in weight of 32 pounds. She felt weak, and consulted her physician, who found glycosuria, and prescribed a diet and insulin. One week prior to her second admission, she became extremely weak, complained of air hunger, and her face took on a reddish-purple color. She was admitted to the medical service on April 16, 1936, twenty-nine days after her discharge from the surgical ward, and forty-three days after the accident.

She was then in deep coma, had Küssmaul breathing and marked acetone odor of the breath. Her temperature was 99, pulse 120, respiration 44, and blood pressure 86/48. A catheterized specimen of urine showed 3 plus albumin, 4 plus sugar, acetone and diacetic acid. The blood sugar was 404 mg. CO_2 combining power was not done.

The treatment consisted in gastric lavage, administration of 6,200 c.c. of saline and 5 per cent glucose by hypodermoclysis and venoclysis, and injection of a total of 435 units of insulin during the night. The acetonuria disappeared in five hours, but the glycosuria persisted. The blood sugar reading the next morning was 626 mg. She developed edema, became cyanotic, and died fourteen hours after admission.

An autopsy was performed by the medical examiner who reported the presence of old, infiltrated blood in the deep layers of the galae. The brain was normal on external examination and showed no evidence of surface injuries. The vessels at the base were normal, the ventricles were normal in outline and clear, and there was no evidence of injury or disease in the fourth ventricle. The chest and abdominal cavities were normal. Microscopic examinations of the organs, including the pancreas revealed no abnormalities. No mention was made of microscopic examinations of the brain. The cause of death was given by the medical examiner as "Diabetic coma; diabetes mellitus; laceration of scalp; history of having been struck by street car; accidental."

Comment. We believe that the patient, because of her obesity and family history might, in time, have developed diabetes, and that these constituted the predisposing factors, while trauma and shock were the precipitating cause of the disease. She had passed a life insurance examination, and repeated urinalyses made by her physician before the accident were negative for sugar. The actual physical trauma and shock, the cerebral concussion and the hemorrhage, must be accepted as proof of traumatic diabetes in this instance.

CASE II. M. T., 14 years old, was admitted to the medical service on January 5, 1938. The family history was negative for diabetes as far back as information was obtainable—the grandparents. At the age of 5, he had sustained a fractured skull and remained in a hospital for four weeks. At the age of 9, he had a second accident with no definite findings, leaving the hospital in good condition after a few weeks. At the age of twelve, he passed a physical examination at school, including urinalysis. Tonsilectomy had been done at 10. There were no other serious illnesses until the summer before admission, when he developed an otitis media which cleared up after two months.

Ten days before admission to the hospital he developed excessive thirst and polyuria and lost 10 pounds in weight. He remained active for about a week, but his mother noted a peculiar flushing of the face. A dose of castor oil given for constipation brought on nausea and vomiting. He became progressively worse and finally stuporous and was brought to the hospital.

On admission he was semicomatose, had acetone odor to the breath, a flushed face, rapid heart, Küssmaul breathing, and a friction rub was heard in the right axilla. (X-ray examination of the chest a few days later was negative.) The blood sugar was 390 mg., and the CO_2 combining power 21.7 per cent. The urine showed 4 plus sugar, acetone and diacetic acid.

Treatment consisted in administration of a total of 418 units of insulin during the first twenty-four hours, combined with intravenous saline and glucose. The diabetes was well controlled at the end of the second week in the hospital and the boy was discharged on February 1, 1938. He was given a diet of 200 Gm. of carbohydrate, 60 of protein and 75 of fat, with 23 units of protamine insulin daily. A sugar tolerance test was made two days before discharge and the following values were obtained: one-half hour 121 mg.; one hour 358; two hours 348; and three hours 272 mg.

Comment. This boy developed diabetes suddenly, after a low-grade infection of two months' duration. He presents a history of trauma on two occasions early in life. There are no predisposing factors, such as obesity or family history. Whether or not trauma played a part in the obscure etiology we cannot say, but it may be assumed that it did. The case is one of many that we encounter, of sudden onset of diabetes with no demonstrable causative factor, and it emphasizes the importance of further study of such cases.

SUMMARY

Two cases of diabetes mellitus are presented, in one of which trauma appears to have been the precipitating cause, in the other a possible predisposing cause. The relationship of trauma to diabetes is discussed, and a plea made for more careful analysis of all cases of diabetes of obscure etiology, and for reports of all cases of diabetes in which trauma has played a part.

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INTRALIGAMENTOUS RUPTURE OF A TUBAL PREGNANCY *

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IT is an accepted assumption that 95 per cent of all ectopic pregnancies are caused by low grade infection of the tubes.[1] The other 5 per cent are caused by tumors, agglutination, angulation, blind ostia, etc. When the passage of the fertilized ovum through the tube is interfered with, imbedding will take place in this structure. The stroma in the fallopian tube is scant and does not always undergo decidual changes to resist the digestive action of the trophoblast of the ovum. Consequently, unless the fetus dies, the pregnancy must terminate by rupture of the tube, or by extrusion of the fetus through the fimbriated end into the peritoneal cavity.

After the ovum imbeds itself, the major portion of the growth may be toward the lumen of the tube (luminal imbedding), or toward the wall of the tube (mural imbedding). Some grow equally in both directions (Falk). Near the fimbriated end of the tube, the luminal type of implantation is most frequent. This type is usually operated on as an "unruptured ectopic" or tubal abortion. As one approaches the cornual end of the tube, the mural or combined type is more frequently encountered. It is here that we see the acute catastrophe of a ruptured tubal pregnancy.

When tubal rupture does occur, it may be intra- or extraperitoneal. By far the most common is the intraperitoneal rupture. The incidence of extraperitoneal rupture, or rupture between the layers of the broad ligament is about 1.4 per cent.[2] This is about one extraperitoneal rupture in seventy-five cases of ruptured tubal pregnancy.

Following the rupture of the tube extraperitoneally, the pregnancy may continue unrecognized until term, and a living fetus may be delivered by operation; the fetus may die, with retention of the gestation sac within the layers of the broad ligament for varying lengths of time. Lastly, with rupture of the tube, there may be immediate death of the fetus, with formation of a hematoma in the layers of the broad ligament. This may dissect upward, giving a large retroperitoneal hematoma, or it may rupture secondarily into the peritoneal cavity.[2]

The first intraligamentous rupture of a tubal pregnancy reported by the Harlem Hospital Gynecological Service was seen in April, 1932. The second, which we report now, was seen in March, 1937. During this five year period 149 ectopics were admitted to the service.

J. H., aged 24, was admitted on March 31, 1937, with a complaint of vaginal bleeding of six weeks' duration. Dysuria, frequency, and urgency were noted for four weeks. The patient began to bleed on February 20, which was the expected date for menstruation. The period began normally, stopped after three days, and then started again. Very mild bleeding was noted until the date of admission, with some increase of the amount of flow during the three days in March when the patient expected her period. No cramps, chills, fever, nausea, vomiting or syncope were noted. No previous menorrhagia, metrorrhagia, or dysmenorrhea was reported. The patient had had one child.

General physical examination was essentially negative. Abdominal examination showed slight tenderness in the hypogastrium. Pelvic examination revealed a 2 finger introitus, firm perineum, and a rather roomy vagina. The tip of

the cervix was softened, the os closed. There was no discoloration. The uterus was anteflexed and slightly enlarged. Hegar's sign was positive. The right fornix was free and non-tender. High up in the left fornix, there was a globular, tender mass, about 4 cm. in diameter. There was slight blood staining of the gloved finger.

Red blood cells numbered 3,900,000, with 70 per cent hemoglobin. Leucocytes were 5,800, with polys 46 per cent, transitionals 6 per cent, lymphocytes 48 per cent. The sedimentation rate was 18 mm. in one hour. A 2 plus Kahn test was reported. An Aschheim-Zondek test was done, but the rabbit died. A second rabbit was not available before operation.

The patient remained on the ward under observation for fourteen days. Bleeding ceased, The abdomen was soft, there was no tenderness, the temperature was normal, and the patient was almost symptom-free. However, she was operated on with the preoperative diagnosis of left ovarian cyst, chronic salpingitis, and left tubal pregnancy to be excluded.

The abdomen was opened through a left paramedian incision. There was no free blood in the peritoneal cavity. The intestines were walled off with laparotomy pads. The right tube and ovary appeared normal, the uterus slightly enlarged. In the region of the left adnexa, there was a large, tortuous, cystic, tense mass, filling the left pelvis. It was deep bluish in color with dilated blood vessels over its surface. The medial end of this cystic blended with the cornual end of the tube. The cystic mass was followed laterally to what appeared to be the fimbriated end of the left tube. It was then noted that the two layers of the broad ligament had been dissected by the dilated left tube. The left ovary was not seen, but it was assumed that it was included in this mass. Adhesions were freed, and during the manipulation the mass was ruptured, spilling forth several blood clots, a live fetus (about three months' size) and a placenta.

The cornual end of the left tube was clamped and cut. The left infundibulopelvic and left broad ligaments were doubly clamped and excised from the uterus by means of a wedged-shaped incision. The wound in the uterus was closed by means of a figure-of-eight suture. All bleeding was controlled. The appendix was not investigated. The peritoneum was closed with a continuous suture, and a one point suspension done at the lower angle of the

peritoneal closure. The abdominal wall was closed in layers. No drains were inserted. The patient was returned to the ward in good condition.

The postoperative course was complicated by a mild thrombophlebitis of the left pelvic veins. The patient was discharged on the twenty-fourth postoperative day in good condition. She has since been entirely well.

Pathologic Examination. The tube was 6 cm. in length, mildly thickened and tortuous. The ampullary portion measured 1.7 cm. in diameter, and was perforated along its entire length. Spongy friable tissue adhered to the mucosal surface of the tube in the perforated areas. The mesosalpinx and broad ligament showed moderate hemorrhagic discoloration. The fetus was 7.5 cm. long, attached to the placenta by a numbilical cord measuring 10 cm. The placenta was $6\frac{1}{2} \times 4$ cm. and showed small areas of gray and red infarction.

Ruptured ampullar gestation with chronic pseudofollicular salpingitis was the final diagnosis.

COMMENT

It is of interest to note that both cases of intraligamentous rupture of a tubal pregnancy reported by the Harlem Hospital occurred on the left side. Of interest also is the fact that the patient attended to her usual duties as a domestic up until the time of admission, and the only reason she entered the hospital was because of mild, prolonged, vaginal bleeding.

CONCLUSIONS

1. The diagnosis of extraperitoneal rupture should be considered when any tentative diagnosis of tubal pregnancy is made, even if the patient presents mild symptomatology.

2. The alteration of the menstrual cycle is a most important symptom in the diagnosis of ectopic gestation.

3. The findings on pelvic examination may be misleading.

4. The Aschheim-Zondek test may or may not aid in the diagnosis.

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CARBUNCLE OF THE KIDNEY

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THE first description of a carbuncle of the kidney was given by Israel[1] in 1894. The lesion which he noted in the kidney was similar in appearance to a carbuncle on the neck from which his patient had been suffering. Because of the similarity, this observer gave the name, "renal carbuncle," to this pathologic entity.

Moore[2] defines carbuncle of the kidney as a metastatic, circumscribed, conglomerate, suppurative process usually caused by the Staphylococcus aureus, and having its origin in some superficial focus, such as a furuncle, paronychia or carbuncle. The lesion is very frequently limited to the cortex and does not involve the pelvis of the kidney. The typical carbuncle is composed of many small suppurating areas and the entire lesion is separated from the kidney parenchyma by a ring of inflammatory tissue.

It appears that not enough emphasis has been placed upon this condition. There are some who still confuse it with other suppurative conditions of the kidney, or else do not recognize this lesion in the kidney. The acute septic or embolic kidney which shows numerous small abscesses must be separated from the carbuncle, and, in fact, presents a different pathologic picture. There is another condition which must not be confused with carbuncle. This is a metastatic inflammatory lesion found in the cortex of the kidney, caused by the staphylococcus, but which does not show multiple points of suppuration. It may have a necrotic center and an inflammatory tissue reacting wall about it. Many writers have classed these with carbuncles, but some, such as Smirow,[3] call these single, metastatic, staphylococcal abscesses of the kidney cortex, and definitely separate them from carbuncles.

It is generally agreed upon that car-
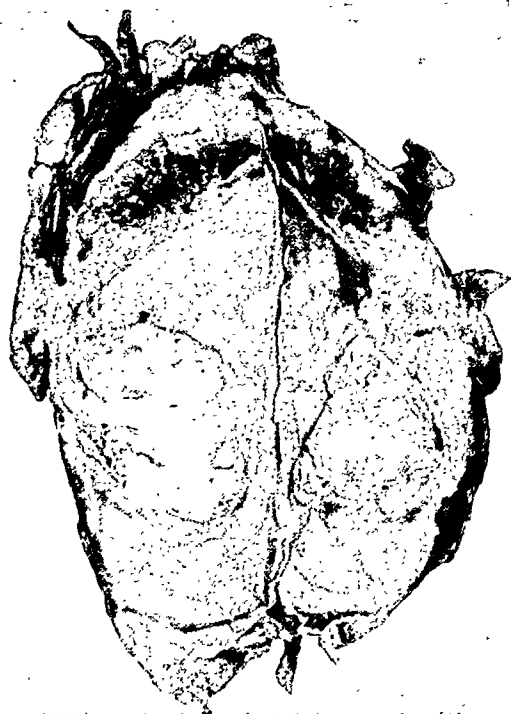
FIG. 1. Carbuncle of the kidney showing numerous suppurative foci with the thickened necrotic capsule visible at the lower pole.

buncle of the kidney is a metastatic lesion from some focus elsewhere in the body. Just how this lesion forms once the organisms arrive in the kidney, is not definitely settled. Some writers believe that a septic embolus is wedged at the point of branching of a small artery, where it is gradually broken down into smaller pieces containing many bacteria. These, then, are carried to different portions of a small area of the kidney with the subsequent formation of many small adjoining abscesses, called together a carbuncle. Barth's[4] conception of the pathogenesis, however, is that a few organisms will settle at one point, multiply

and by spreading along the lymphatics will produce multiple suppurative areas.

Carbuncles of the kidney are usually unilateral. A few cases of bilateral lesions have been reported. Israel[5] and other investigators explain the unilaterality as due to a lowered resistance of one of the kidneys which has occurred because of injury or previous disease. Schnitzler[6] and Brewer[7] have shown the importance of injury in the production of these lesions. Hunner[8] has demonstrated the rôle which incomplete kidney drainage plays in kidney disease and this may further account for the unilateral occurrence of carbuncle of the kidney.

Renal carbuncles may frequently form perinephritic abscesses. Some investigators believe that all perinephritic abscesses are secondary to this condition. Brady[9] points out that it is characteristic that a carbuncle does not involve the pelvis of the kidney. He states that if the infection has ruptured into the pelvis, the condition is no longer a carbuncle or cortical abscess but a pyelonephritis.

CASE REPORT

J. M., a white male, aged 18 years, entered the hospital on May 14, 1938 with the history of fever and pain in the left side. The pain, which began one week before admission, was at first diffuse over the entire back and then became localized to the left lumbar region. Three days before admission he developed malaise and a fever of a 102°F. There was no history of chills but the patient did complain of nausea and vomiting. One week before the onset of symptoms, the patient stated that he had had a small abscess over the lateral border of his left wrist which was opened and had healed. The past surgical, medical and venereal history was negative.

Physical examination revealed a well developed and well nourished white male, who appeared acutely ill. The temperature was 101.5°F., the pulse 110 and the respirations 26. The face was flushed, the throat congested. The heart was not enlarged and no murmurs were heard. The abdomen was flat and no masses were palpable. Tenderness and spasm were noted over the left kidney region, but the liver, spleen and kidneys were not palpable. Rectal examination was negative.

Laboratory Findings. The urine was cloudy; reaction acid; specific gravity 1.010; albumin, sugar and microscopic examinations were negative. The blood chemistry showed a urea of 16.8 mg. and a glucose of 111 mg. per 100 c.c. of blood. The blood Wassermann was negative. P.S.P. showed an excretion of 85 per cent in two hours. The white blood count was 12,500 with a differential of 82 polymorphonuclear leucocytes and 18 lymphocytes. A pyelogram was done which revealed a definite anterior displacement of the left kidney on lateral view. There was no obliteration of the kidney outline or psoas shadow. Cystoscopic examination revealed a normal urinary bladder mucosa. Excretion from both kidneys was good and approximately equal on both sides. A K.U.B. film showed no abnormalities.

Clinical Course. The preoperative diagnosis was abscess of the left kidney with perinephritic extension. A nephrectomy was performed. Following the operation, a wound infection developed and the temperature varied between 99° and 104°F. Transfusions and infusions were given. The patient gradually gained strength, the wound healed slowly and he recovered completely, five weeks after admission.

Pathologic Report. The specimen consisted of a kidney (Fig. 1) which measured 11 × 6 × 4 cm. The capsule throughout, especially at the lower pole, was markedly thickened, hemorrhagic and necrotic in areas. The cut surface revealed a mass measuring 4.5 cm. in diameter. It was located in the cortex and the medulla of the kidney. This mass was fairly well circumscribed and yellowish brown in color. A small amount of pus could be expressed from the cut surface. Culture from this pus was positive for Staphylococcus aureus. The surface of the mass had a mottled appearance and numerous small necrotic yellow areas could be seen throughout. The rest of the kidney appeared grossly normal.

Microscopic examination of the mass showed almost complete destruction of the kidney parenchyma by an inflammatory reaction. Here and there the remains of a few glomeruli and tubules were seen. Immediately surrounding the mass the tubules were flattened and many were filled with neutrophiles. The intertubular tissues in this region contained numerous fibroblasts, neutrophiles and many markedly congested blood vessels.

DISCUSSION

The clinical diagnosis of renal carbuncle presents great difficulties and as a matter

of fact is not often recognized. There is usually a history of some superficial infection with the subsequent development of fever, pain and definite tenderness in one flank. Physical examination may show some muscular rigidity in the upper abdominal and lumbar muscles with moderate but not well localized tenderness. The kidney may or may not be palpable. Leucocytosis is the rule and frequently some abnormality in the urine such as a few red blood cells and a moderate amount of albumin is present. Blood cultures may be positive. Cystoscopic examination may reveal some infection on the affected side with diminution of renal function as compared with the opposite side. The pyelogram may show displacement of the kidney, compression of the pelvis or other filling defects. Intravenous urography may occasionally aid in the diagnosis. Another laboratory test has been found useful. Often, and more so on the left side, due to the extension of the infection to the diaphragm, fixation or limited motion of the diaphragm will occur. X-ray or fluoroscopic examinations will allow the examiner to view this phenomenon.

The gross appearance of the lesion is that of a localized necrotic mass within the kidney parenchyma which is of an inflammatory origin. Multiple foci of infection which are demarcated by a fibroblastic wall or which fade gradually into the surrounding tissue without a limiting membrane, may be seen. Abscess formation is late and when it occurs the lesion is preferably referred to as an abscess. The carbuncle is usually located near one of the poles of the kidney and is localized more to the cortex than to the medulla. The process tends to move towards the capsule rather than the pelvis of the kidney.

This is a very serious disease. The sooner the diagnosis is made and treatment instituted, the better the prognosis. The treatment is surgery, which may consist of one of several procedures, such as incision and drainage followed later by nephrectomy, if necessary, primary nephrectomy or excision of the carbuncle. The choice of procedure depends upon the condition of the patient. The lowest mortality appears to be where enucleation is performed but the cases in which this may be done are few. Nephrectomy, in general, appears to be the operation of choice, providing the condition of the patient permits it. When a perinephritic abscess is present, incision and drainage are indicated. Recovery may follow this procedure of drainage without any other treatment being necessary. It is also likely that some carbuncles are never diagnosed and recover spontaneously.

SUMMARY

A case of renal carbuncle with recovery following nephrectomy is reported.

Carbuncle of the kidney is a definite pathologic entity and should be so recognized.

The gross appearance is similar to that of a carbuncle found on the superficial portions of the body.

It must not be confused with other suppurative conditions of the kidney.

The diagnosis is difficult but if considered, especially when the condition arises following a superficial infection, plus the aid of certain laboratory procedures, it may be more frequently recognized.

The disease is a serious one. The treatment is surgery, the procedure being either incision and drainage, nephrectomy, or excision of the carbuncle. The sooner treatment is instituted, the better the prognosis.

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MALIGNANT TUMOR OF ADRENAL CORTEX

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KNOWLEDGE concerning adrenal tumors is constantly increasing. In the interest of presenting another example of malignant growth of the adrenal gland this case report is submitted.

CASE REPORT

Mrs. I. S., age 30, was sent to the Syracuse Memorial Hospital, October 12, 1937. The patient complained of pain in the left upper abdomen, backache and scanty menses. She had been comparatively well until January, 1937, at which time there was a marked lessening of menstruation, a general lack of endurance, increase of weight and an abnormal growth of hair on the face and body.

The patient was married and has one child. Her menses began at the age of 16 and had always been regular, lasting four to five days with a moderate flow. On close questioning the patient stated that the periods may have been shortened earlier. The January period lasted less than two days and no period since had been of greater duration. One menstruation was markedly delayed although no entire month was skipped. Her last period was just before admission to the hospital and lasted only part of one day. The patient has gained between 20 and 25 pounds during one year, and now weighs 193. Her face and body had become covered with hair, enough on her face so that she had been obliged to shave. Her sex desires were normal up to about eight months before, but since January, 1937 there had been an absence of libido. The patient had become nervous and listless.

Examination revealed a markedly overnourished adult female, short and thick set, with coarse facial features. There was an acne-like eruption on the skin of the face, with a rather profuse growth of hair on the upper lip and chin. The voice was coarse and somewhat masculine. The neck was thick and short with no enlargement of the thyroid. The breasts were large and pendulous, the abdomen prominent with a large fat pad overhanging the pubis. The hair on the pubis assumed masculine distribution. A large mass was palpable in the upper left abdominal quadrant, extending over to the midline and down below the crest of the ilium, moving on deep inspiration. There was generalized left-sided tenderness on deep pressure.

The external genitalia were not markedly abnormal, nor was the clitoris hypertrophied. The uterus was normal in size but more firm and fibrous than the uterus of the average woman of 30.

Hypertrichosis of the arms and legs was noted.

The blood pressure was 136/112. The blood count showed 5,000,000 red cells with 80 per cent hemoglobin, leucocytes 8,300, with 83 per cent polys and 17 per cent lymphocytes. The urine on October 12 was negative, except for albumin 1 plus. On the 19th the urine showed 4 plus sugar. A glucose tolerance test was not done. The Wassermann was negative. The N.P.N. was 30 mg. and the blood sugar 93 mg. per cent. Basal metabolism was minus 5 The sedimentation time was 63 minutes.

Cystoscopic examination revealed slight congestion about the left ureteral orifice with a delayed function of the left kidney. A pyelogram revealed the left kidney to be low in position, apparently due to a mass above pressing it downward. X-ray examination of the chest was negative. A diagnosis of tumor of the left adrenal was made.

Operation. A Cabot incision was made in the left upper quadrant from the xiphoid down the midline nearly to the umbilicus and then laterally to the costal margin. The flap was reflected upward and the abdomen explored. The gall-bladder was soft and the pelvic organs appeared normal. The right kidney was normal and there was no apparent enlargement of the right suprarenal.

The tumor lay in the left upper quadrant with the spleen freely movable above and lateral to it. The left kidney was eventually found below and unattached to it. The tumor itself was soft and about the size of a football.

It had a yellowish-red mottled appearance with many large blood vessels on its surface. With considerable difficulty the tumor was mobilized and the bleeding was controlled. In delivery the growth ruptured, spilling a small mass of friable material into the retroperitoneal space. The tumor was removed and the area drained after closure of the incision in the mesentery of the colon. Throughout the operation the patient received intravenous saline and glucose and at the termination, a transfusion of 500 c.c. of citrated blood was given. The patient returned to her room in good condition.

Twenty-four hours postoperatively, the patient's temperature suddenly rose to 105 with the pulse at 140 and respirations 30. The blood pressure dropped to 95/60, and a few hours later it was 65/40. She had cold, clammy extremities, an ashen appearance and a rapid, thready pulse. Adrenalin was given in ½ c.c. doses every hour for four hours. Caffeine and strychnine were also given as well as fluid by subcutaneous injections. The patient improved under this treatment and was able to take nourishment the following day. Adrenalin was given by hypodermic every four hours for the next forty-eight, at the end of which time she had improved enough to discontinue it. The symptoms of severe shock which she exhibited are very common following the removal of cortical adrenal tumor and many patients succumb at this point. The shock in this case was probably due to postoperative adrenal failure from which the patient recovered. She continued to improve from this time onward and left the hospital on the twenty-seventh day postoperatively, apparently in very satisfactory condition.

Pathologic Report. The specimen consisted of a large tumor measuring 19 × 14 × 11 cm. Portions were encapsulated by a thin membrane, which contained a considerable number of blood vessels. The contents extruded from all parts of the specimen but the gross appearance was generally the same throughout. The tumor was soft, friable and broken up into innumerable fine projections. When the mass was bisected there was a definite pattern resembling a cauliflower-like material with a yellow stem. The medullary portion had a distinctly yellow color and was firm in the outer portions, with the center soft, friable, almost necrotic. The outer portion had the appearance of brain tissue.

The tumor cell tended to have rather clear or finely granular cytoplasm, resembling adrenal cortex cells. The arrangement was in rather solid masses with very little evidence of alveolar structure.

Tumor cells were separated from numerous blood spaces by very thin walls or endothelium. Hemorrhage and necrosis were very prominent factors. Mitotic figures were fairly numerous. Microscopically, it was a malignant tumor of the hypernephroma type.

The patient was last seen five months postoperatively. There was no significant weight change. The hair on the face had completely disappeared and that of the abdomen and legs was practically normal in distribution and amount. She had menstruated twice since leaving the hospital and periods were longer but as yet not normal. There was definite pallor, and the red cell determination was 3,600,000, the hemoglobin 60 per cent (Tallquist). There was no evidence at that time of a recurrence of the growth.

In February, 1939 a local recurrence was noted for the first time. This extended rapidly and the patient died six months later.

COMMENT

The patient who is the subject of this report presented as definitive symptoms of her adrenal pathology the hypertrichosis and masculine escutcheon, coarseness of the skin, increase in weight (20 to 25 pounds in one year) oligomenorrhea, loss of sexual desire and a large abdominal mass. The severe shock which appeared twenty-four hours postoperatively was presumably due to temporary adrenal insufficiency, often observed among postoperative sequelae in this type of case.

A UNIVERSAL ADJUSTABLE STIRRUP FOR IMPROVED LITHOTOMY POSITION[*]

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OPERATIVE procedures on the perineum require a position which will insure adequate exposure so that

FIG. 1. Stirrup apparatus applied to operating table. Either knee or foot suspension is readily available.

the work can be accomplished with reasonable ease and therefore with a safer and better technique. With the common type of stirrup supplied with most operating tables a sufficient exposure can sometimes be obtained, but not infrequently this proves impossible due to difference between (a) end-width of the operating table and perineal width of the patient, and (b) thigh and leg length measurements in relation to any fixed height of suspension.

In 1907 Young wrote of the importance of a proper position for operative exposure of the perineum for prostatic work, and at that time used the Halsted board with upright stakes. Since then he has devised and described his special table with stirrups for this improved position. Cecil designed stirrups to fit the standard operating table and uses the head of the table to raise the perineum. He discarded the Halsted board in view of the flexibility of the modern operating table equipped with correct stirrups. Because of the difficulty of maintaining a good position for children undergoing cystoscopic examination and eliminating assistants to hold the patient, Campbell fashioned stirrups to fit the standard office examining table for this purpose.

The new type of stirrup here presented may not be superior to the extremity-suspension apparatus described by these authors, for their instruments appear to fulfill the requirements for which they were designed. However, this "universal" stirrup possesses some different features which lend themselves to the various conditions encountered so that the same stirrup may be employed in all instances of lithotomy position.

Description of Stirrup Instrument. The instrument is made of a steel alloy which will withstand without bending or turning pressure exerted either by lock devices of

the table or from muscular efforts of patients during incomplete anesthesia.

The complete stirrup apparatus attached child or adult. One end of rod D is threaded into the inner end of bar C. The free end of rod D is for connection of the stirrup
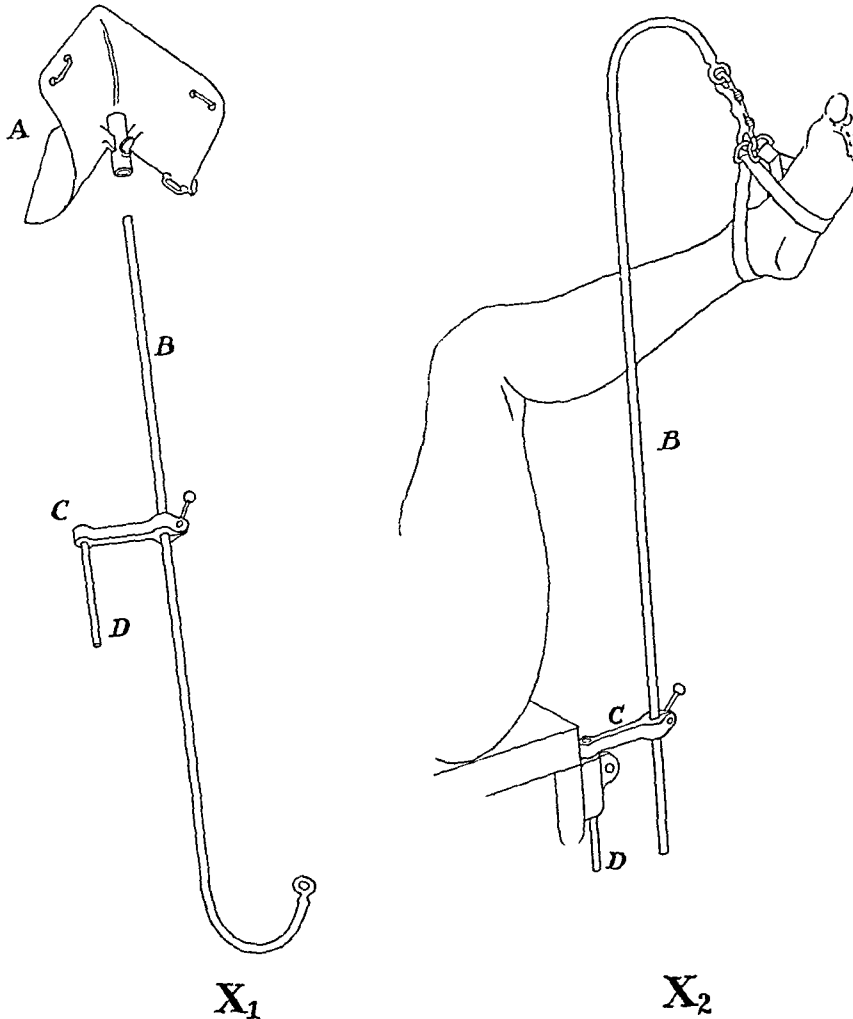
FIG. 2. Component parts of the stirrup apparatus. X_1. A, knee crutch may be attached to straight end of rod B; horizontal bar C contains slit clamp at one end through which rod B passes, while into the other end is threaded rod D for attachment to operating table. Rod B is 31 inches long, bar C is 5 inches long and 1 inch thick, rod D is 7 inches long. X_2. Stirrup apparatus applied to table for ankle-foot suspension. Buckles and canvas strapping are attached to the cane shaped end of rod B.

to the operating table is shown in Figure 1, and its component parts of the instrument in diagrammatic fashion in Figure 2. According to the type of suspension desired, whether knee or foot, the straight end of rod B is passed up or down through the slit clamp in the outer end of bar C. Thus rod B may be locked positively at any position with the handle of this clamp and may be adjusted to suit the various heights for different patients, be they short or tall,

apparatus to the operating table. It is the only part of the instrument for which specification must be made as to diameter because of the variation in size of slot openings for stirrup attachment in different tables. Since the working distance of bar C is 4 inches on either side, by horizontal adjustment, i.e., locking the stirrup toward or away from the table, the distance between the right and left stirrups may be diminished or increased at any point up to

8 inches. In this manner a wide or narrow perineum may be brought to or over the end of the operating table without undue

FIG. 3. Application of stirrup apparatus for children. Rod D is rotated inward in front of the table end and locked in position to accommodate perineal width. Rod B is lowered and locked at the proper level to accommodate for thigh and leg length.

stretching of thigh and leg and without impingement of the perineum on the vertical rods of the stirrup. (Figs. 3 and 4.)

When there is insufficient room for first and second assistants so that they must stand behind the patient's thighs, as is the case with many stirrups now in use, their view of the operative field is apt to be obstructed, leading to tiring and inaccurate assistance. In this awkward position, particularly when knee crutches are employed, there is a tendency for the assistants to rest or lean on the patient's legs to reach the operative field, with resultant pressure on the nerves and vessels of the popliteal space. These difficulties are eliminated by providing space for the assistants in front of the patient's thighs. (Fig. 5.)

To be prevented from obtaining a moderate or an exaggerated perineal position because the perineum cannot be

FIG. 4. Application of stirrup apparatus for exaggerated perineal position in adults such as is employed in perineal prostatectomy. Rod D is rotated and locked in extreme outward position, thus increasing the width through which a wide perineum may be brought unhindered over the end of the operating table.

FIG. 5. Showing the added valuable space afforded assistants and operator in front of the operative field.

brought to or over the end of the table; to have the patient's feet swing too closely toward each other or close to the operator's face; to work with insecurely locked stirrups, or those that bend or twist so that the perineum is not maintained in the proper position, definitely increases the difficulty in operating and proves generally annoying.

CONCLUSIONS

A new and simple type of stirrup is presented which has the following features:

1. Combined foot and knee suspension without changing stirrup apparatus.

2. Made of steel to withstand encountered pressure.

3. Adjustable to correct heights for the various patients so that it can be used on children as well as on any size of adult.

4. Can be used on any operating or examining table having a slot for stirrup attachment.

5. Makes available valuable space for assistants in front of instead of behind the patient's extremities.

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THE term "shock" should not be used unqualified. In civilian surgery it should be preceded by the word "surgical" or "traumatic" and in military or air raid surgery the term "wound shock" (1917) is accurate and descriptive. This terminology avoids confusing the condition with "shell-shock" or shock resulting from a transient psychical disturbance.
From—"War Wounds and Air Raid Casualties" (Lewis).

A NEW INSTRUMENT FOR PASSING PORTIONS OF TENDONS AND FASCIAE LATAE

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THE technical difficulties frequently encountered in the passage of portions of tendons and fasciae latae through having a spike in its serrated mouth. The diameter of the forceps rod (Fig. 3a) commonly employed at The Mayo Clinic

FIG. 1. Tendon and fascia passer in closed position; a forward movement of the closing handle brings the two blades of the forceps together.

FIG. 2. Tendon and fascia passer with blades of the forceps open to receive material to be passed.
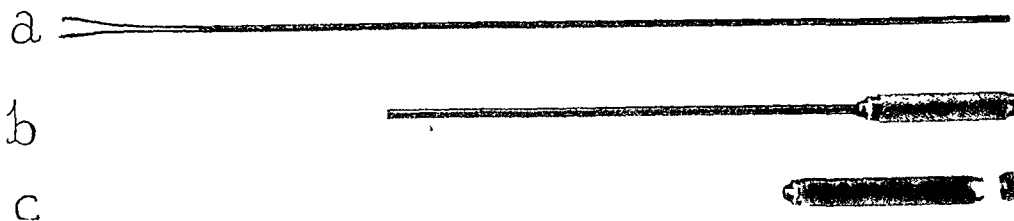
FIG. 3. A, forceps rod; B, tube and handle for closing the blades of the forceps; C, handle with serrated proximal circumference.

bone, stimulated interest in the development of an instrument (Fig. 1) which would facilitate this procedure. The instrument and the technique attendant on its use are particularly applicable to plastic procedures performed in the regions of the shoulder and knee joints, and may be employed further in the transplantation of tendons through soft tissues.

The instrument consists of a tissue forceps (Fig. 2) enclosed by a tube; the forceps possesses an unusually long handle is 0.125 inches (3.17 mm.), the closing tube (Fig. 3b) is 0.135 inches (3.42 mm.) in diameter, and an outside closing tube is 0.191 inches (4.85 mm.) in diameter. There is one handle (Fig. 3c) affixed to the forceps rod of the instrument, and another handle, affixed to the tube, for closing or opening the blades of the forceps. Each of these two handles is 0.37 inches (9.40 mm.) in diameter. The over-all length of the instrument is 12.5 inches (31.75 cm.).

[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

MUSINGS ON THE BILIARY TRACT

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INTEREST in the biliary tract has been manifested since earliest times, beginning apparently in the interpretation of animal sacrifices as a part of divination in which an attempt was made to foretell future events or discover hidden knowledge.

Divination, according to Jastrow,[1] was either voluntary or involuntary. Involuntary divination concerned itself with the understanding of observable facts or events which came to the attention without being deliberately sought. The positions of the stars, the appearances of the clouds and the movements of the planets required some explanation. The birth of a monster and an unusual flight of birds demanded clarification. Much significance was attached to these interpretations.

On the other hand, there was voluntary divination in which one purposely looked for some means by which he might draw aside the curtains and catch a glimpse of an unknown future. In voluntary divination one did not wait to observe in what direction birds were flying or in what formation they were arranged. He did not wait to count their number. Instead, some means at hand was used and an explanation sought. One set loose a flock of birds,

observed the direction in which they went, the formation they assumed and the number of birds which followed a definite path.

In the region of the Tigris and the Euphrates several rather detailed systems of divination were produced. After evil had come and the wrath of the gods had descended, people resorted to incantations. Divination had a more purposeful task. This was a method of attempting to change the course of future events or at least preparing oneself for what was to come. Incantations were curative in design; divinations were preventive.

Three systems of divination were perfected in Babylonia and Assyria. One was the inspection of the liver—hepatoscopy. Another was the observation of signs noted in animals at birth. Especially in this second system, the greatest significance was naturally attached to marked abnormalities. The third system was astrology. The movements of the sun, moon, planets and stars—which were all closely identified with the gods—represented the gods actively preparing events shortly to take place on earth.

The inspection of the liver of a sacrificial animal was perhaps the oldest method of

divination. Even before the heart came to be regarded as the center of mental and emotional activity there was, according to Jastrow,[2] "an earlier period in which that distinction was accorded to the liver." Pliny the Elder[3] (23–79 A.D.) mentions the fact that the heart was not used for the purpose of divination until after the 126th Olympiad (274 B.C.). Plato[4] (427–347 B.C.) in the *Timaeus* stated that "the authors of our being, remembering the command of their father when he bade them create the human race as good as they could, that they might correct our inferior parts and make them to attain a measure of truth, placed in the liver the seat of divination."

Why was the liver singled out to be the favorite organ of revelation? Jastrow held that the explanation was a very simple one. Because of the great vascularity of the liver and because blood was associated with life, many primitive peoples considered the liver the center of all mental and emotional activity. It was the seat of life; more than that, however, it was the seat of the soul. Before a journey, a building operation or a military expedition was begun, by the aid of hepatoscopy an attempt was made to gain insight into the future.

Since the gall-bladder was always considered an integral part of the liver because of its very close relationship, it was particularly observed. Peculiarities as to shape, size and position of the gall-bladder were noted in detail. Much attention was directed to the markings on the liver made by the gall-bladder and bile ducts. Observers recalled some past experiences when favorable or unfavorable events followed certain findings. The essential character of the viscus was also considered. Past experience and the association of ideas—these principles determined the interpretations which were made.

What were some of these interpretations? A large, swollen gall-bladder indicated there would be an extension of power; a small and shrunken gall-bladder presaged weakness. In the elaborate system of interpretation which had been developed a

sign on the right side always related to the king; a sign on the left side, to the enemy. If the right side of the gall-bladder were tightly embedded in its fossa, surely the king's army would increase in strength. If the gall-bladder were tightly adherent on the right side but loose on the left, it was a favorable sign for the enemy. This observation signified that the king's army would be held in a tight and close grasp. If the left side of the gall-bladder were tightly adherent the indication was that the enemy would be kept as a prisoner and the king's army would be victorious or, at least, the enemy would be kept under control. If the left side showed a tightly embedded viscus, the enemy would be victorious and would hold firmly the king's army in its grasp.

If the common hepatic duct were securely located within the porta hepatis, success would be assured; one not within such protective confines meant an open front to the enemy. A long common bile duct was a most desirable portent because it foretold a long life. The surface markings made by the bile ducts were especially noted by the diviners, and the anomalous conditions which they encountered lent great variety to their interpretations.

Hepatoscopy naturally led to the anatomical study of the liver. Besides the various lobes, the portal vein and porta hepatis, especial interest centered in the gall-bladder and the markings made by the bile ducts. Scribes recorded the interpretations assigned to these variations. Clay models of the sheep's liver were prepared and used for instruction by those who taught in the temples. A picture of such a model now in the British Museum and dating from the time of Hammurabi (2000 B.C.) has been published in "Cuneiform Texts from Babylonian Tablets, etc., in the British Museum." This model is covered with cuneiform writing. It was, in reality, a diagram to explain an omen text. The gall-bladder, portal fissure, cystic duct and part of the hepatic duct are clearly indicated. The gall-bladder, appropriately

enough, bore the name of "the bitter part." The hepatic duct was called "the outlet"; the common bile duct, "the yoke." The porta hepatis was designated as "the crucible."

Almost the same system of hepatoscopy was in practice among the Etruscans. In the Museo Civico at Piacenza is a bronze model of a sheep's liver which dates from the third century B.C. Jastrow was particularly interested in the similarity of this bronze liver of Etruria to the Babylonian counterpart of earlier date which has been described. In the writings of the Greeks and Romans incidental notice is made of Greek and Roman hepatoscopy. There were collections of signs and interpretations of these signs in Roman handbooks but they have not been preserved.

In early zoological literature the gall-bladder in animals was discussed. Aristotle[5] (384–322 B.C.) called attention to the fact that some animals possess a gall-bladder and some do not, and that "among viviparous quadrupeds the stag has none, nor the deer, horse, mule, ass, seal and some swine." Aristotle mentioned the Achainian stag "which appears to have the gall in the tail" and remarked further that "that which they call gall in these animals resembles it in color, but it is not liquid like gall, but more like the spleen in its internal structure."

Pliny[6] was aware of the fact that not every animal had a gall-bladder. He recorded that "at Chalcis, in Euboea, none of the cattle have it, while in the cattle of the Isle of Naxos, it is of extraordinary size, and double, so that to a stranger either of these would appear as good as a prodigy." To Aristotle's list of animals which had no gall-bladder he added the camel and dolphin. He further commented that "some kinds of rats and mice have it. Some few men are without it, and such persons enjoy robust health and a long life." Galen[7] (131–201), the "prince of physicians," had noticed that there were animals in which there was no gall-bladder.

Observations on the gall-bladder have had historical associations as well as those of a religious and scientific nature. Illingworth[8] relates a story which has been told concerning Alexander the Great (336–323 B.C.), the son of Philip of Macedon. When Alexander and his troops returned to Babylon from India, he learned that the sheep which had been sacrificed in honor of his return had no gall-bladder. Alexander considered this a serious omen. In his retreat from India dysentery had taken a heavy toll among his soldiery and he himself was exhausted. Within a few days Alexander also was dead.

Pliny the Elder[9] records an experience of the Emperor Augustus. In the year 32 B.C. the Roman triumvirate came to an end and the Senate declared war on Cleopatra. In 31 the opposing forces met at Actium. Octavian led the land forces; Agrippa commanded the navy. The morning of the battle the Emperor Augustus found a double gall-bladder in the animal he was sacrificing. Great meaning was attached to this discovery, and the overpowering victory of the Roman troops that day made the double gall-bladder seem especially significant to them.

In classical literature one finds references concerning the biliary tract. In the *Electra* of Euripides (480–406 B.C.), Aegisthus, with Pylades for a helper in his task, inspected the viscera of a sacrificed calf. The attention paid to the meaning of the sacrifice is realized as one reads (lines 826–829):

"Aegisthus grasped the inwards
And gazed thereon. No lobe the liver had;
The gate-vein, the gall-bladder nigh thereto,
Portended perilous scathe to him that looked."

It will be recalled that in the *Prometheus Bound* of Aeschylus (389–314 B.C.) the punishment meted to Prometheus was that his liver was to be gnawed upon by a vulture. No more heinous punishment could have been devised in those days. These lines (493–496) are also of interest:

"Smoothness of inwards of beasts sacrificed,
Their hue, which are well-pleasing to the gods,
The gall, the liver's dappled shapeliness—
I set forth the significance of all."

It is not only in the literature of ancient Greece that we find such references. From Chaucer's *Former Age* we recall (line 47) that "Hir hertes were al oon without galles." The absence of the gall-bladder in the pigeon was known to Shakespeare for in *Hamlet* (Act II, scene 2, lines 612–613) the Prince of Denmark himself declares:

"But I am pigeon-liver'd, and lack gall
To make oppression bitter."

The growing realization of the importance of diseases of the biliary tract has been a dominant feature of medical progress in recent years. The methods of study and their far-reaching results are new, but the interest which has developed in this field is only a renewal of one of man's earliest experiences in anatomical observation.

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THE ESTIMATION

OF

PELVIC CAPACITY

BY

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PREFACE

THE present monograph is essentially a collection of ideas concerning the female bony pelvis in its relation to childbearing and, more particularly, of the estimation by pelvimetric methods of its capacity for that function. The application of modern roentgenometric methods to pelvimetry has created a widespread interest in the subject, which has served to emphasize the pioneer work of such men as Michaelis, Litzmann, Breus, Kolisko, and Whitridge Williams of our own time.

The author's interest in this field now extends to a score of years during which time he has written various communications on this subject. A large part of the material here presented has appeared in these writings and credit should, therefore, be given to certain publications in which it has appeared in part. These are *The American Journal of Surgery, Journal of the American Medical Association, American Journal of Obstetrics and Gynecology, Surgery, Gynecology and Obstetrics, Yale Journal of Biology and Medicine*, and the author's monograph *The Obstetric Pelvis*. Mention should be made of the courtesy extended by the publishers of the last two publications for permission to reproduce certain illustrations. The author is gratified at this opportunity to record his appreciation of the continued interest and coöperation in his work of his colleagues, Dr. A. H. Morse, Dr. H. M. Wilson, and their associates. Finally, mention should be made of the generous support of the author's work over a period of years by the Research Funds of the School of Medicine, Yale University.

H. T.

THE ESTIMATION OF PELVIC CAPACITY

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INTRODUCTION

"Very many be the perils, dangers and throngs which chance to women in their labour." These words, written in 1634 by Thomas Raynalde, the author of the first obstetrical treatise in English, are not out of date, for today, three centuries later, the physician is still seeking for ways to lessen the hazards of childbearing. The dangers to which this early obstetrical author referred, like those of the present day, group themselves into three general categories; namely, those associated with abnormalities of the pregnant state, with puerperal complications, and with mechanical difficulties encountered at the time of parturition. We readily recognize the splendid progress that modern scientific medicine has made toward the solution of these problems, particularly those associated with the first two groups, and during the last decade with the aid of roentgenology new information has given us knowledge which may be applied to the solving of many of the problems associated with the last named group.

Experienced obstetricians will readily agree with Rongy that "Obstetrics is largely a mechanical art. Every case of labor is an engineering problem. The obstetrician, like the engineer, must guide himself wholly in accordance with the principles which make a given mechanical problem safe or unsafe, possible or impossible." Furthermore, it becomes apparent that a knowledge of the contours and dimensions of the bony pelvis is the base upon which the attack on these mechanical problems must be built. We should recall, however, that the successful process of parturition depends not only upon the size and conformation of the bony birth canal, but upon such coördinating factors as the size and position of the fetus, the force and action of the uterine contractions, and the adaptability of the pelvic soft parts.

It is the purpose of this monograph to outline methods which may be employed in ascertaining the contours and dimensions of the bony pelvis in the living subject and to point out the findings that may be expected from such investigation.

"Fortunate it would be indeed for childbearing women" wrote Ramsbotham in 1855, "if they each possessed a pelvis of the figure and dimensions already given as standard. Such, however, is by no means the case; and this organ is subject to great varieties, as well in form as size. It would indeed be difficult to select from all the preserved specimens in existence any two which exactly resemble each other." At a somewhat later date Sir William Turner wrote "with the exception of the skull no portion of the skeleton presents greater individual variations than the pelvis." Roentgenologic studies in our own day have amply proved the soundness of these observations and have further demonstrated that at times apparently minor pelvic variations have a profound effect upon the course and character of labor. In most instances minor pelvic variations do not form insuperable obstacles to labor, but their importance as a factor in labor pathology must not be minimized. There is no question that a large proportion of untimely and ill-advised obstetrical operations are the direct result of a lack of proper understanding of the mechanical aspects of labor in which variations in the contours and the dimensions of the pelvis may play a major part.

Some idea as to the wide range of variability which is found at the pelvic inlet in white women of our population may be gained from the results of a study of 800 adults measured roentgenologically at the Yale Clinic. In 16.1 per cent of this series the transverse diameter was actually shorter than the anteroposterior in this plane. In 45.9 per cent the transverse diameter was no more than 1 cm. longer than the anteroposterior. In 34.0 per cent the transverse diameter was from 1 to 3 cm. longer than the anteroposterior. Finally, in 4.0 per cent this diameter was 3 cm. or more longer than the anteroposterior. As with the pelvic inlet, so also the midpelvis and pelvic outlet show wide ranges in variation. It is obvious, therefore, that if we possess accurate information respecting the size and configuration of these various portions of the pelvis, we shall have an understanding which will be greatly useful in treating many of the problems associated with labor.

It is now nearly a score of years since the author began to study pelves roentgenologically, and so impressive has been the usefulness of the information so obtained that, almost from the beginning, he has recommended the use of Roentgen pelvimetry as a routine procedure in primigravidae. The soundness of this recommendation is being proved with the years of experience, and it is significant that a leader like Munro Kerr within the present year has recorded his

views as follows: "I do not think that, with the knowledge now at hand regarding the influence excited by even minor variations of pelvic formation, we who advocate routine pelvic radiography for all
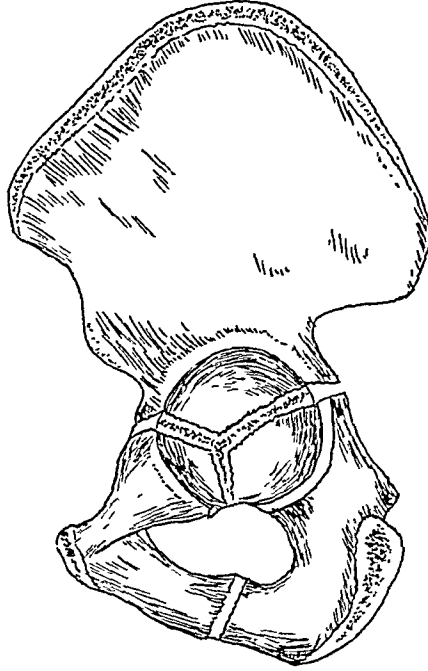
FIG. 1. The three primary centers of ossification unite at the acetabulum about the time of puberty. (From Thoms' "The Obstetric Pelvis," William & Wilkins.)

primigravidae have lost our sense of proportion in this matter, as many suggest today. On the contrary, I make bold to predict that before many years pass it will be accepted as an essential detail of the antenatal examination."

CONSIDERATIONS OF THE BONY PELVIS

Before mensuration of the pelvis can be discussed, the bony pelvis must be considered in itself. Our knowledge of this structure has been greatly increased during the last decade, but is by no means complete, as is witnessed by the numerous investigations which are now being conducted in this field. In our own clinic male as well as female pelves are being studied in subjects of all ages, ranging from infancy to adulthood, in an effort to learn not only information concerning the incidence of pelvic variations, but something of the factors which combine in their production. The not infrequent occurrence of male pelvic characters in female pelves is a fact which has considerable obstetric significance and which will be discussed later.

It is apparent that there are two sets of factors in operation in the evolution of the pelvis: (1) developmental and hereditary influences; and (2) influences the result of mechanical factors. In the fetal pelvis, long before mechanical factors have been brought into play, sexual differences are readily apparent. At birth the pelvis is both cartilaginous and bony, the innominate bone being in three parts—ilium, ischium and pubis—which are united by cartilage at the acetabulum. The acetabula, iliac crests and ischial rami are almost entirely cartilaginous in their structure. The various parts of the acetabulum are not completely united until after puberty. The pubic and ischial rami unite at about the eighth year. At puberty the secondary centers of the crest of the ilium, the anterior inferior iliac spine (Fig. 1), the symphysis pubis and ischial tuberosity appear, but these structures may not be fused until the twentieth year. Thus it will be seen that during skeletal growth the pelvis is in a relatively plastic state and it is evident that mechanical influences must play an important part in its eventual architecture.

Among these influences may be mentioned the effect of muscular action, the body weight, and the upward and inward force of the heads of the femora. When abnormal plasticity of the bone is present, as in rickets, it is easy to see that marked deformity may thus occur. Schultz, whose studies in primates are fundamentally important, calls attention "to the evident close connection between mechanical factors and evolutionary changes in the primate pelvis, a connection which exists nowhere more pronouncedly than in the pelvis of man."

In considering the capacity of the birth canal it should be remembered that its structure is not only bony but also fibrous and muscular. Furthermore, the bony pelvis is not a definitely fixed structure. Its articulations, which are apparently fixed by strong ligaments, undergo a certain amount of softening under the influence of pregnancy, giving a certain degree of mobility to the pelvic bones. Thus, at the end of pregnancy strong flexion of the thighs on the abdomen, either in the exaggerated lithotomy or squatting positions, definitely, if but slightly, enlarges the pelvic outlet. The well-known Walcher position, where forced extension of the thighs is present, gives a slight enlargement to the pelvic inlet. However, this latter position is so difficult to maintain that its use in dystocia problems is so limited as to be of little practical use. The symphyseal joint separates to a greater or less extent under the influence of pregnancy. At full term this spread averaged 0.58 cm. in thirty-four cases studied by

the author. Rarely the separation may be so marked as to incapacitate the patient, as in the author's reported case. However, the softening of this and other pelvic articulations usually causes but
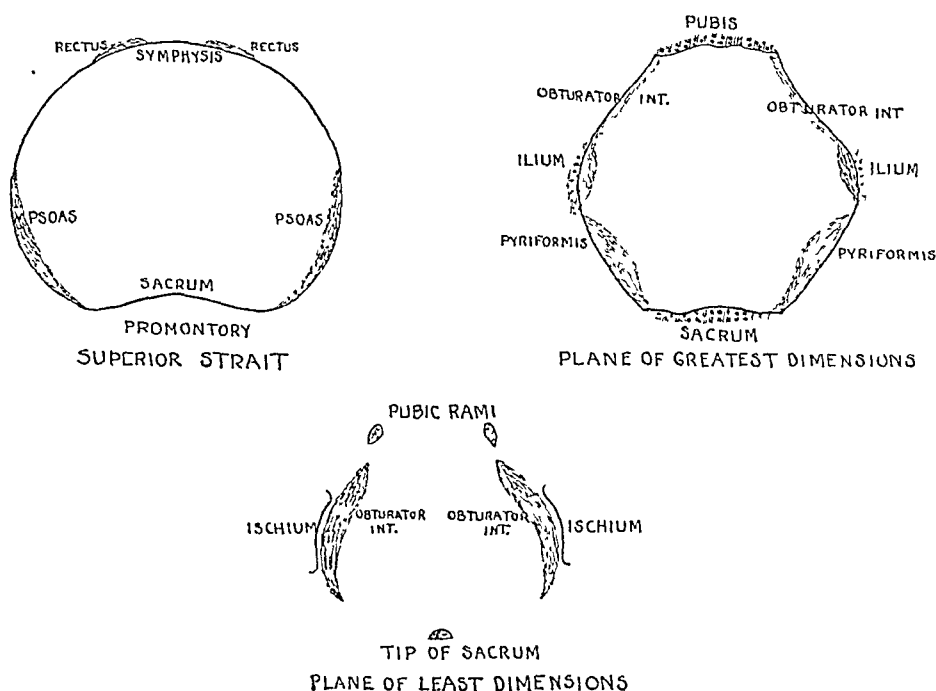
FIG. 2. Diagram showing encroachment of soft parts at different levels of the pelvis. (From Thoms' "The Obstetric Pelvis," Williams & Wilkins.)

little actual enlargement of the pelvis, although in some cases delivery is probably somewhat facilitated.

Certain of the pelvic soft parts may encroach more or less upon the pelvic cavity. In the region of the pelvic inlet the psoas and iliacus muscles protrude slightly as they pass over the pelvic brim (Fig. 2); the rectum if distended may also decrease somewhat pelvic capacity. In the midpelvis the obturator internus and pyriformis project slightly. In the lower pelvis the structures making up the pelvic floor form a barrier at the lower pole of the pelvic canal until this is overcome by the processes of labor. It is, therefore, correct that when we speak of the estimation of pelvic capacity in its true sense we should consider not only bony conformation but these soft structures. However, it is the more or less unyielding bony pelvis which is the chief concern of the obstetrician in estimating pelvic capacity.

The importance of an understanding of the sexual differences in the bony pelvis has been referred to. As early as the third month in fetal life male and female pelves may be differentiated. In the male

fetal pelvis the entire cavity has a more funnel-shaped appearance than in the female, and the sacrosciatic notch in the former is narrower and deeper. According to Thompson "It appears that during
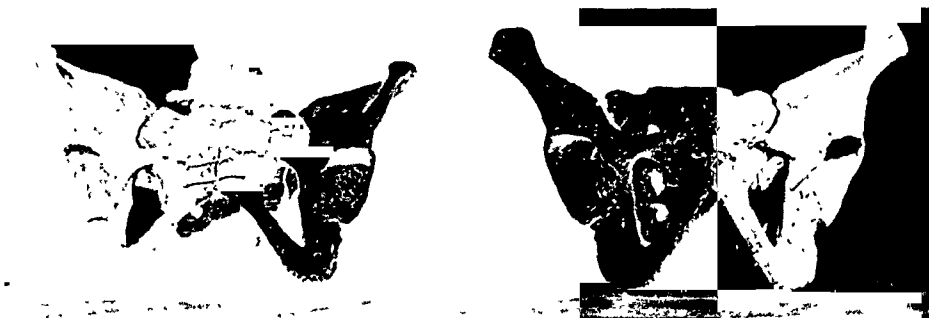
FIG. 3. Front view female and male pelvis showing characteristic differences in pelvic arch.

fetal life the essential sexual characters are as well defined as they are in adult forms, and that any differences that occur during growth between the adult and fetal forms, due it may be to the influence of pressure or muscular traction, affect both sexes alike, and that such influences are in no way accountable, as has been maintained, for the characteristic features of the pelvis as contracted with the male."

In the adult pelvis the sexual differences may be conveniently tabulated as follows:

Female	Male
Greater size, lighter build	Heavier, rougher, more massive bones
Greater capacity of pelvic inlet	Lessened capacity of pelvic inlet
Greater width of pubic arch	Pubic arch narrower, more angular
Larger interischial diameter	Side walls converge, forming a deeper pelvis
Lessened convergence of side walls of cavity	
Obturator foramen ovoid	Triangular obturator foramen
Great sacrosciatic notch, large, almost, rectangular, with longer periphery	Smaller sacrosciatic notch boundaries meet above at acute angle
Ilium larger than male	
Posterior boundary of sacrosciatic notch chiefly sacral	Posterior boundary of notch sacral only in lower part

Certain characters of the male pelvis are of definite obstetrical interest because of their occasional occurrence in female pelves. These are: (1) the long narrow sacrosciatic notch and the sacrum in a more forward position which lessens the space between the lower anterior surface of this bone and the ischial spines; (2) the narrow subpubic angle caused by the narrowing course of the pubic rami; and (3) the narrowing of the pelvic side walls from above downward, which restricts pelvic width especially in the mid and lower pelvis. (Figs. 3 and 4.)

The shape of the male pelvic inlet has been described in textbooks almost universally as essentially "heart-shaped" with narrow fore-part or retropubic angle and, in comparison with the female, a

FIG. 4. Lateral view female and male pelvis showing characteristic differ-ences in the sacrosciatic notch and position of sacrum.

posteriorly displaced transverse diameter. These characters have been referred to as typical of the male pelvic inlet and to be quite typical of the "android" pelvis. However, the results of recent investigations by W. W. Greulich and the author do not agree with this concept. (Fig. 5.)

In a Roentgen study of this plane in sixty-nine adult white males of the medical student group, we have found the same essential pelvic variations in pelvic inlet contour which we have found in females, and our conclusions are here repeated.

1. The pelvic bones are heavier than those found in females and, in general, the whole pelvis has a more angular appearance.

2. The pelvic inlet in general appears more circular, and the posterior sagittal diameter in this plane is slightly shorter than that seen in female pelves. This is due to a slight displacement poste-riorly of the widest transverse diameter. The forepart of the pelvic inlet differs but slightly from that seen in female pelves and the so-called "heart-shaped" pelvis was not characteristic of the great majority in our series.

3. The constant characters seen were the structural heaviness and prominence of the ischial spines with narrowing of the pelvic side walls from above downward, the angular pelvic arch with rela-tively narrow subpubic angle and the type of sacrosciatic notch which has been described as characteristically male.

[7]
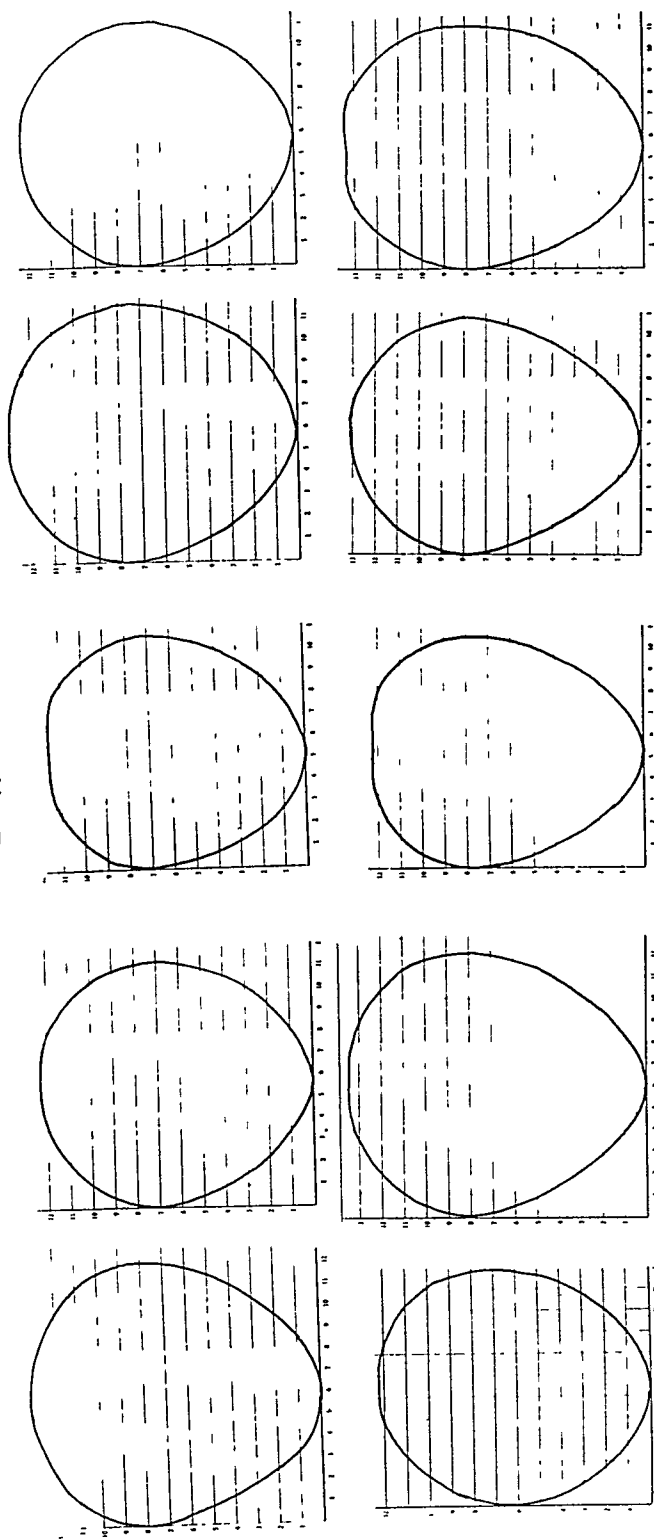
GROUP 1

FIG. 5. Specimen tracings of pelvic inlet contours of male pelves showing dolichopellic, mesatipellic and brachypellic types.

[8]

GROUP 2

FIG. 5. (Continued)

[9]
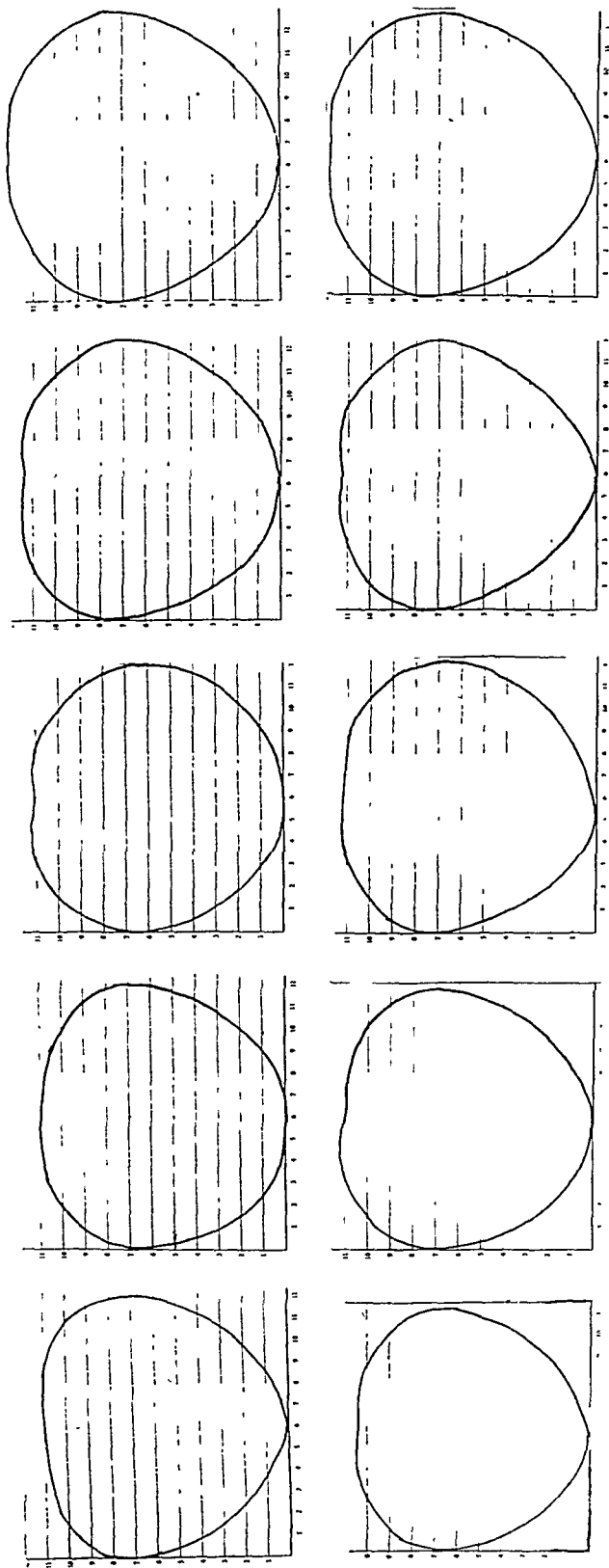
4. The three main pelvic types which we have found in female pelves, i.e., dolichopellic, mesatipellic, and brachypellic were also found to occur in male pelves.

In the foregoing we have discussed the essential differences between male and female pelves, and it is of the highest importance that these facts should be thoroughly understood if we are to intelligently study the obstetrical significance of pelvic capacity.

It is important next to consider the planes and diameters of the bony pelvis which are significant from the point of view of practical obstetrics.

THE SIGNIFICANT PELVIC DIAMETERS

From the point of view of practical obstetrics there are three portions of the pelvis the mensuration of which will yield useful information. These are: (1) the plane of the pelvic inlet; (2) the midpelvic plane in the lower midpelvis; and (3) the planes of the outlet. A knowledge of the various diameters of these planes will not only furnish a concept of the architecture of the pelvis, but will also give an adequate index of the available space present in the bony birth canal. In addition, this knowledge will enable us to incorporate in the prenatal history a useful and instructive record.

The Plane of the Pelvic Inlet. From the obstetrical point of view this plane is bounded anteriorly by the posterior upper surface of the symphysis pubis and the forward portions of the iliopectineal lines, laterally by the iliopectineal lines, and posteriorly by the posterior portions of these lines and the anterior upper surface of the sacrum at the point where the convergence of these lines takes place. It should be noted that this plane is not that of the anatomic superior strait, but rests slightly below this. Its importance in the mechanism of labor has been emphasized, particularly by Caldwell, Moloy, and D'Esopo. In order to simplify the subject, this plane might also be called the obstetrical inlet of the pelvis. The anteroposterior diameter of this plane is referred to by DeLee as the second conjugate. The useful diameters of this plane are (Fig. 6): (1) anteroposterior, (2) transverse and (3) posterior sagittal.

1. The *anteroposterior diameter* of the pelvic inlet extends from a point on the upper posterior surface of the symphysis about 1 cm. from the superior border, posteriorly to the anterior surface of the first sacral vertebra at the point where the iliopectineal lines would

meet if they were to be continued. (This point of convergence may or may not be located at the sacral promontory. It is usually somewhat below this eminence.)

Fig. 5. (Continued)

GROUP 3
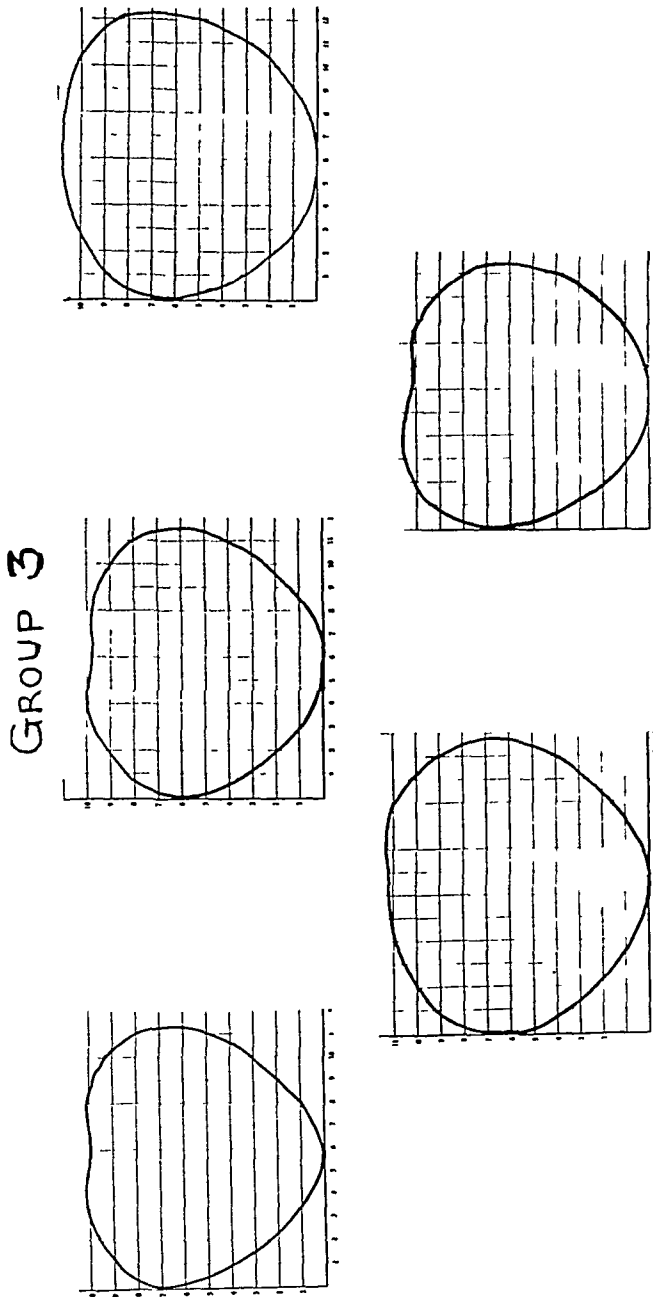
2. The *transverse diameter* of this plane is the widest transverse distance separating the iliopectineal lines. It bisects the anteroposterior somewhat posterior to its midpoint, dependent upon the shape of the plane of the pelvic inlet.

3. The *posterior sagittal diameter* of the inlet is represented by that portion of the anteroposterior diameter which lies posterior to the point of intersection by the transverse diameter. The length of
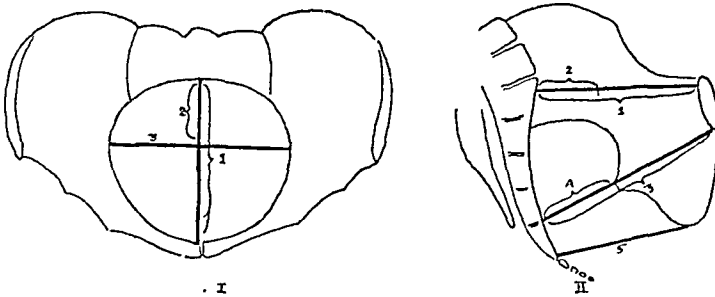
FIG. 6. Significant diameters of the pelvis. *I*. Diameters of the pelvic
inlet: 1, anteroposterior diameter. 2, posterior sagittal diameter.
3, transverse diameter.

II. Diameters of the lateral pelvis: 1, anteroposterior diameter
of inlet. 2, posterior sagittal diameter of inlet. 3, anteroposterior
diameter of midplane. 4, posterior sagittal diameter of midplane.
5, posterior sagittal diameter of outlet. (From Thoms and Wilson,
in *Yale J. Biol. & Med.*, 11: 179, 1939.)

this diameter is an index of the amount of space in the important upper posterior pelvis and when abnormally shortened usually represents a posterior displacement of the transverse diameter. This may be due to an abnormal convergence of the iliopectineal lines anteriorly, or, in the case of the flat pelvis, to a definite antero-posterior shortening of the inlet of the pelvis. If due to the latter, the discrepancy between the lengths of the anteroposterior and transverse diameters is at once apparent.

The Midpelvic Plane. The second obstetrical plane is the mid-pelvic plane. It is somewhat ovoid in form, large anteriorly and small posteriorly, being narrowed posteriorly by the convergence of the sa-crosciatic ligaments. This plane has been defined as being bounded anteriorly by the lower border of the symphysis pubis, laterally by the ischial spines, and posteriorly by the tip of the sacrum. However, as Hanson has emphasized, these points are not truly in the same plane, for the tip of the sacrum is usually as much as 2 cm. below the level of a line joining the lower border of the symphysis with the bispinous diameter. Therefore, the posterior limit should be placed in the lower third of the sacrum and will usually find itself at the junction of the fourth and fifth sacral segments. In our studies of the lateral pelvis aspect, the necessity for this concept of the midpelvic plane has been repeatedly emphasized.

The three diameters which are of obstetrical significance in this plane are the transverse or bispinous, the anteroposterior, and the posterior sagittal.Th is latter diameter, as in the case with the plane
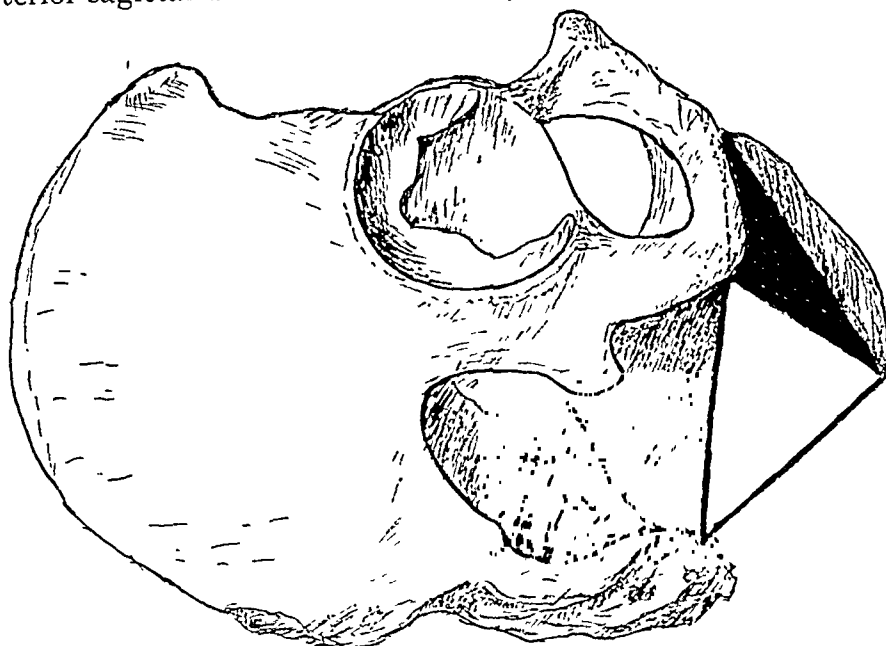
FIG. 7. The planes of the pelvic outlet are essentially two triangles whose bases meet on a line drawn between the tips of the two ischial tuberosities. (From Thoms' "The Obstetric Pelvis," Williams & Wilkins.)

of the pelvic inlet, represents that distance on the anteroposterior diameter which lies posterior to the point of intersection by the transverse or bispinous diameter.

Planes of the Outlet. The third portion of the pelvis of definite obstetrical significance is the pelvic outlet. This is in reality two planes—represented by two triangles, the bases of which join each other along the line of the bituberal diameter. (Fig. 7.) The anterior triangle is bounded by the bones forming the pubic arch, the shape of which is of considerable obstetric significance. The two diameters of particular significance are the transverse of the outlet or bituberal diameter, and the posterior sagittal. This latter diameter extends from a midpoint on the bituberal diameter posteriorly to the tip of the sacrum. Other diameters for the pelvic outlet have been described, but do not have essential obstetrical significance and for routine purposes may be omitted.

In addition to the above diameters, all of which may be determined satisfactorily by simple Roentgen methods, there are certain measurements that may be determined by manual methods. These are:

1. The interspinous diameter: The distance between the outer surfaces of the iliac spines.

2. The intercristal diameter: The widest distance between the outer lips of the iliac crests.

3. The external conjugate or Baudelocque's diameter: The distance between the anterior surface of the pubis and the depression under the last lumbar spine.

4. The intertuberal or transverse diameter of the outlet: The distance between the lower innermost surfaces of the ischial tuberosities.

5. The diagonal conjugate diameter: The distance between the sacral promontory and the inferior surface of the symphysis pubis.

The latter two measurements will be considered in more detail later, but the first three have in recent years been shown to be less reliable than was formerly supposed. In 1933, in a paper entitled "The Inadequacy of External Pelvic Measurements," the author compared the results in a Roentgen study of seventy-five pelves. It was pointed out that external measurements must be regarded chiefly as guide posts, for while it was true that small pelves as a rule were accompanied by small external measurements, the reverse was certainly not the case. For instance, in two cases of the series the external conjugate diameter was 20.0 and 16.0 cm. respectively, yet the anteroposterior diameter of the inlet was 10.0 cm. in each. Again in two subjects with identical external conjugate diameters (17.0 cm.) the anteroposterior of the inlet was 7.75 and 11.0 cm. respectively. Students of this subject are therefore in agreement with DeLee in his statement that external measurements are unreliable indices of the size of the pelvic cavity. Of some significance, however, may be the relationship of these three diameters to each other in the case of rachitic pelvis. Possibly for this reason, if for no other, they should be retained.

In addition to the above diameters, which are of great usefulness in determining pelvic capacity, we should mention certain pelvic contours which are also of importance. The shape of the pelvic inlet, the vertical and lateral contours of the anterior sacral surface, the shape of the sacrosciatic notch, the convergence of the pelvic side walls and the prominence of the ischial spines, and the character of the pubic arch are all considerations which should be taken into account. We shall consider these points in greater detail later.

To recapitulate—we can say that the significant diameters for use in the routine survey of the bony pelvis are:

Pelvic inlet......................................
$\begin{cases} \text{Anteroposterior} \\ \text{Transverse} \\ \text{Posterior sagittal} \end{cases}$

Midpelvic plane...........................
$\begin{cases} \text{Anteroposterior} \\ \text{Transverse or bispinous} \\ \text{Posterior sagittal} \end{cases}$

Pelvic outlet.............................
$\begin{cases} \text{Transverse or bituberal} \\ \text{Posterior sagittal} \end{cases}$

The above measurements may be considered the cardinal diameters of the pelvis. Of less significance are the diameters known as:

Interspinous
Intercristal
External conjugate or Baudelocque
Diagonal conjugate.

PELVIC VARIATIONS

From what has been said it is obvious that variations in form of the bony structures of the pelvis show wide distribution. Because of this it has become apparent to all recent students of this subject that the description of the "normal female pelvis" as noted in most modern textbooks requires considerable revision. It is not difficult to understand how an error of this nature has persisted when we consider the relatively limited material which was available to observers in previous times. The use of roentgenologic methods of investigation allows an extensive use of material and it is significant that the occurrence of this textbook type of female pelvis is notable by its relative infrequency. At the present time in white women in this country it is probable that what has previously been described as the normal type of female pelvic inlet occurs in less than one-third of all women.

The intermingling of sex characters and the variability of form of the female pelvis is so great that attempts at classification become most difficult. Some idea of this difficulty may be gained from the statement of Caldwell and Moloy to the effect that "it is difficult to find a pelvis which is typically female in all proportions." The work of these authors in the field is extensive and noteworthy, and the classification which they have suggested, based largely on sex characters, is ingenious and undoubtedly useful for anthropologic study. It is, however, not of easy clinical adaptation because of its complexity. The authors admit that the intermingling of types

renders difficult any attempt to gain simplicity in this method of classification.

It appears to the writer that for the sake of clarity and for direct
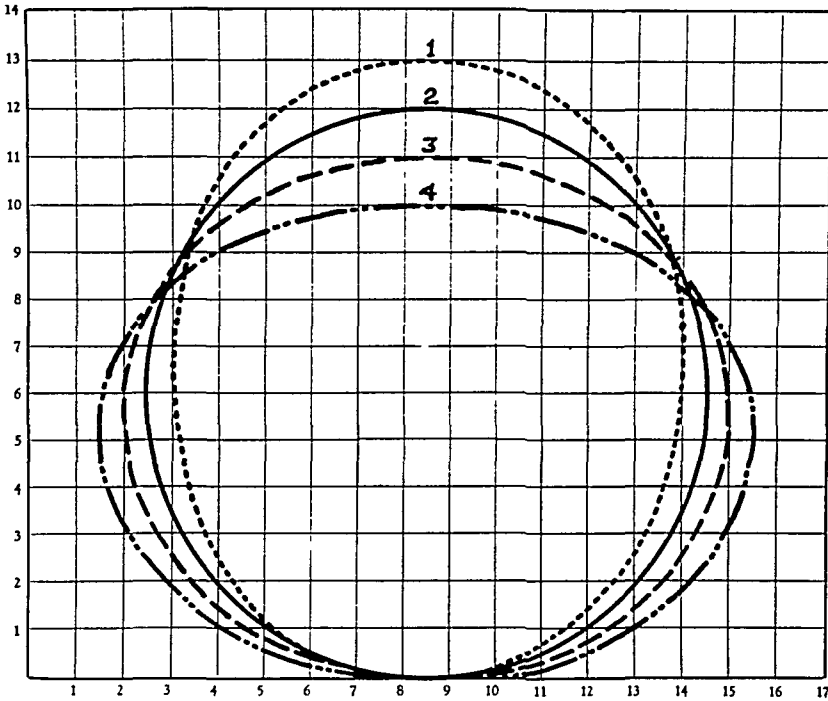
FIG. 8. See text.

clinical application a classification based upon the *general shape* of the pelvic inlet has many advantages. For the past four years we have used such a classification in our clinic and its use by members of our staff over that period has testified to its adaptability to routine work. When it is considered that during this period we have measured the pelvis roentgenologically in every primigravid woman who has been delivered in our clinic, it can be seen that its application has not been inconsiderable.

We consider, therefore, that in general the pelvic inlet may assume one of four shapes (Fig. 8), which may be described as: (1) an anteroposterior oval; (2) a circle; (3) a transverse oval; and (4) an exaggerated transverse oval. We can further describe this morphology by considering the relation of the anteroposterior and transverse diameters of these figures to each other, and on this basis we may adopt terms for pelvic types which will be useful:

1. Dolichopellic or anthropoid type pelvis. The transverse is less than the anteroposterior diameter.

2. Mesatipellic or round type pelvis. The transverse is equal to, or slightly greater (not more than 1 cm.) than, the anteroposterior diameter.

3. Brachypellic or oval type pelvis. The transverse diameter is more than 1 and less than 3 cm. greater than the anteroposterior diameter.

4. Platypellic or flat type pelvis. The transverse diameter is 3 cm. or more greater than the anteroposterior diameter.

The above classification is useful simply as a basis for description of individual pelves; beyond this it does not pretend to venture. After all, in the individual case the obstetrician is not so much concerned with the group in which a pelvis may align itself as he is with the measurements and contours which reveal its useful capacity for childbearing. Given the eight cardinal diameters which have been described, together with a brief consideration of the important contours, such as inlet, notch and pubic arch, one can form a fairly adequate estimation of pelvic capacity for obstetrical purposes.

It might be suggested that we could more correctly use the above terms describing inlet relationships if they were based upon the pelvic index (the ratio of the anteroposterior diameter to the transverse diameter × 100) rather than upon the arbitrary figures chosen. For certain statistical purposes this may be so, but for useful clinical adaptation the use of the index as a basis for classification is cumbersome and does not accomplish any useful purpose. It has also been suggested that the above terms based upon the interrelationships of two diameters may not give a true idea of the shape of the pelvic inlet. This is entirely conceivable but, given the pelvic type plus the knowledge of the three cardinal diameters of this plane, such objection is not well sustained. In any attempt at classification we must bear in mind that if we are to escape being lost among the trees we must keep a perception of the forest ever in mind. With this idea in mind let us recall our original statement that *pelves in general* may be divided into four groups, those whose inlet approximates (1) an anteroposterior oval; (2) a circle; (3) a transverse oval; (4) an exaggerated transverse oval.

It is important to review some of the characteristics of male pelves which may affect female pelves:

1. The male characteristics which may affect the pelvic inlet are: (a) The inlet is in general more circular. (b) The widest transverse

diameter is, therefore, slightly displaced posteriorly. (c) The posterior sagittal diameter is relatively shortened.

2. The male characteristics which may affect the midplane are: (a) The narrowing of the pelvic side walls with increase in size and prominence of ischial spines results in a relatively decreased bispinous diameter. (b) Laterally the male type or narrow sacrosciatic notch is present which decreases posterior pelvic capacity in this plane and manifests itself in a relatively shortened posterior sagittal diameter.

3. The male characteristics which may affect the pelvic outlet are: (a) Narrowing of the pubic arch is present, with less arcuate formation of its sides and a relative shortening of the bituberal diameter. (b) Because of the forward displacement of the sacrum forming the posterior wall of the male notch, the posterior pelvic capacity in this plane is also decreased, which manifests itself in a relatively shortened posterior sagittal diameter of the outlet.

These male characters may appear in female pelves at the inlet, the midpelvis, or the outlet, and in a certain small group of pelves they may occur in all three portions, the complete male pelvic type. It is obvious that they may have a considerable obstetrical significance, particularly in the latter instance.

Some idea of the distribution of these basic types in white women of the clinic group may be gained from their incidence in 800 primigravidae who were delivered at term (child 2500 Gm. or over) in the New Haven Hospital. (Table I.) Again the fact impresses

TABLE I

Type	Number	Per Cent
Dolichopellic	129	16.12
Mesatipellic	367	45.88
Brachypellic	272	34.00
Platypellic	32	4.00

us that 62 per cent of this series present pelves whose inlet is not that described as normal in textbooks of anatomy. In a recent study by Greulich and Thoms of the pelvic inlet in 789 white females, which included 582 clinic women, 100 nurses, and 107 young females from 5 to 15 years, the incidence of pelvic types was as indicated in Table II. The incidence of the various types in the student nurses

seems especially significant in view of the superior physical status and economic level of the women comprising that group. The data from the younger girls is also of significant interest, showing the high incidence of the dolichopellic type in this group.

TABLE II

	100 Nurses	582 Clinic women	107 Children
Dolichopellic................................	37.0	14.5	57.9
Mesatipellic................................	46.0	44.5	33.6
Brachypellic................................	17.0	34.3	8.3
Platypellic................................	....	6.7	

Our conclusions follow:

1. The type of pelvis which for the past two centuries has been considered normal for white women was found in less than 15 per cent of 582 primiparous clinic patients, and in only 6 per cent of 100 young women from a much more privileged economic group. It was, therefore, neither the normal pelvis—in the sense of being the most frequently occurring type—nor was it the most adequate type, as gauged by the relative frequency of operative intervention required during labor by the women possessing it.

2. It has long been known that marked anteroposterior flattening of the adult pelvis may result from severe rickets during early life. The high incidence of round and of anteroposteriorly elongated pelves among the student nurses of our series suggest the possibility that adequate nutrition during early life and other factors which make for the attainment of maximum normal body size prevent that degree of anteroposterior flattening of the pelvis, which has come to be considered as characteristically feminine.

In the series of 800 white clinic women from whom the statistics quoted above were taken, the entire pelvis was not surveyed roentgenologically because the first part of the group antedated our practice of taking routine lateral views. However, in the latter 200 of this series this was done and in this group the incidence of pelvic type followed the same general trend as in the larger group. (Table III.)

In order to gain some idea of the normal range of measurements, the mean values of the various diameters are given. (Table IV.) From a survey of this table certain conclusions may be drawn. In the dolichopellic type there is a tendency for the anteroposterior-

transverse relationship to be maintained throughout the pelvis. In the brachypellic type a similar tendency is maintained, although in a less striking manner. In the platypellic group the shortened posterior sagittal diameter of the inlet and wide bispinous diameter suggest that rickets plays a rôle in the etiology of this group and evidences of the disease in a distortion of the contours and position of the sacrum will be found in many instances.

TABLE III

Type	Number	Per Cent
Dolichopellic	37	18 5
Mesatipellic	95	47 5
Brachypellic	62	31 0
Platypellic	6	3 0

TABLE IV

	Inlet		Midplane				Outlet	
	A.P.	Trans	P.S.	A P.	Trans	P.S.	Trans	P.S
Dolichopellic	12 53	11 72	5 07	12 55	9 45	5 22	8 95	7 84
Mesatipellic	11 75	12 32	4 48	12 34	10 34	5 23	9 16	7 71
Brachypellic	11 06	12 67	4 15	12 01	10 32	5 23	8 92	8 05
Platypellic	9 00	12 67	2 75	11 67	10 45	4 71	9 12	7 58

For purposes of clinical comparison it may be useful to establish criteria for the designation of *small pelves* in each group. Such a subdivision is necessarily arbitrary. However, if "average" pelves may be defined as those lying within the interquartile range, that is in the middle half of all, then small pelves may be defined as those which are measurably smaller. If we use the anteroposterior diameter of the inlet as the yardstick, we find that for the first three pelvic types the lower limit of the average group is 0.5 cm. less than the mean for the type. About one-fifth of all pelves are measurably smaller and may, therefore, be designated as small. This being so, small pelves occur with approximately equal frequency in the three pelvic types, as indicated in Table v.

It may be clinically useful to study the mean measurements of this group of 200 pelves in white women and discuss the lower limits of normal for each measurement. By this means a table may be made giving us these limits of which we can say that dimensions

smaller than those given for each diameter should be viewed circumspectly. (Table vi.)

TABLE V

Type	Anteroposterior Diameter	Number	Per Cent
Dolichopellic......................	Less than 12 cm.	7	18.9
Mesatipellic........................	Less than 11.25 cm.	17	17.9
Brachypellic........................	Less than 10.5 cm.	11	17.7

TABLE VI

	Inlet			Midpelvis			Outlet	
	A.P.	Trans.	P.S.	A.P.	Trans.	P.S.	Trans.	P.S.
Dolichopellic...........	12.0	11.25	4.5	12.0	9.25	5.0	8.5	7.5
Mesatipellic...........	11.25	11.75	4.0	11.75	9.75	5.0	8.5	7.5
Brachypellic...........	10.75	12.25	3.75	11.5	10.0	5.0	8.5	7.5

Clinical Relationships. In a recent paper the author has analyzed certain clinical relationships of the pelvic variations of the above group of 200 pelves and some of these statistics are of considerable interest. Thus in this group of primigravid women consecutively delivered at term (child 2500 Gm. or over) the labor was terminated by operative intervention thirty-six times, or 18.0 per cent. An analysis of this intervention is:

Dolichopellic Type

1. Small pelvis—contracted midplane and outlet—mid forceps.
2. Small pelvis—contracted midplane—low forceps.
3. Child 3920 Gm. arrest at outlet—low forceps.
4. Child 4175 Gm. arrest at outlet—low forceps.
5. Prolonged labor—contracted outlet—low forceps.
6. Persistent posterior position—low forceps.
7. Prolonged labor—low forceps.
8. Small pelvis—contracted midplane and outlet—low forceps.

Mesatipellic Type

1. Persistent face presentation—cesarean section.
2. Premature separation of placenta—cesarean section.
3. Posterior arrest in midpelvis—mid forceps.
4. Contracted midplane—mid forceps.
5. Child 4180 Gm.—low forceps.
6. Arrest at outlet—low forceps.
7. Small pelvis—prolonged labor—low forceps.

8. Arrest at outlet—low forceps.
9. Small pelvis—low forceps.
10. Prolonged labor—low forceps.
11. Contracted midplane—low forceps.
12. Persistent posterior—low forceps.
13. Small pelvis—low forceps.
14. Outlet contraction—low forceps.

Brachypellic Type

1. Hypertension—dystocia syndrome—cesarean section.
2. Premature separation of placenta—cesarean section.
3. Contracted midplane—child 3935 Gm.—mid forceps.
4. Transverse arrest—contracted outlet—mid forceps.
5. Small pelvis—contracted midplane—mid forceps.
6. Prolonged second stage—child 3980 Gm.—mid forceps.
7. Contracted midpelvis and outlet—child 4135 Gm.—mid forceps.
8. Rheumatic heart disease—mid forceps.
9. Arrest in midpelvis—inertia—mid forceps.
10. Inertia—low forceps.
11. Contracted midpelvis—low forceps.
12. Contracted outlet—low forceps.
13. Small pelvis—contracted outlet—low forceps.
14. Contracted midplane—low forceps.
15. Pulmonary tuberculosis—low forceps.

Platypellic Type

1. Rachitic pelvis—cesarean section.

Omitting outlet or low forceps in this series of 200 deliveries, we find that operative intervention was done in fifteen instances, or 7.5 per cent of cases. These were distributed as follows:

Dolichopellic type—1 operation—2.5 per cent.
Mesatipellic type—4 operations—4.2 per cent.
Brachypellic type—9 operations—14.5 per cent.
Platypellic type—1 operation—16.6 per cent.

These latter figures correspond very well with the operative intervention in a series of 600 cases of delivery in white women at term which were previously reported, and serve to emphasize further our conviction that the most favorable type of pelvic inlet is that which is round or is elongated anteroposteriorly (mesatipellic or dolichopellic) and not the oval (brachypellic) or "textbook" type of pelvis.

In this series of 200 deliveries labor was prolonged (24 hours or more) in thirty-eight instances (19.0 per cent). In eleven of these cases the outlet was contracted, in fourteen the inlet showed dimensions less than normal for the type, and in five the midplane showed contraction, either alone or in combination with some other portion of the pelvis. All of these findings show the importance of an accurate

pelvimetric survey during the antenatal period, and make it apparent that unless primigravid patients are routinely thus examined, many significant changes in the bony pelvis will remain undiscovered.

The subject of pelvic variations is a particularly fascinating subject, not only because this newer knowledge has opened up a wide field for investigation, but also because of the importance of this knowledge to the function of childbearing. It is obvious that if a mammalian species is to survive, the pelves of the females of that species must be large enough to permit the birth of their young. Because of such relationship, anthropologists of the nineteenth century were led to expect a rather close correspondence between the shape of the head and the shape of the pelvic inlet in various races. And today among certain obstetricians there is an opinion that rather close relationships exist between pelvic type and body build. In a recent paper entitled "A Study of Pelvic Type," Greulich and Thoms have reported the results of a study which attempted to investigate such relationships. We measured and photographed 132 of the clinic women and 104 student nurses, all of the latter being college graduates. Some of the relationships between pelvic type and body build in the series are here summarized.

1. The clinic women and student nurses who had dolichopellic and mesatipellic pelves were on the average the tallest women of our series, those with a relatively wider pelvic inlet, the brachypellic type, were the shortest.

2. The clinic women had relatively longer trunks and shorter legs than the student nurses of corresponding pelvic type.

3. The women with long oval pelves (dolichopellic) were predominantly tall, long-headed and broad-shouldered. The width of their pelves between the iliac crests and of their hips between the trochanters was smallest in proportion to the width of their shoulders in comparison with women of the other pelvic types, and they had the largest external conjugate diameter.

4. The women with the transversely elongated pelves (brachypellic) were, on the average, the shortest of the series, and they had the broadest heads, the narrowest shoulders, the widest pelves and hips in proportion to the width of their shoulders and the smallest average external conjugate diameter.

5. The women with round pelves (mesatipellic) were approximately intermediate between the other two groups in all these dimensions.

6. There was much variation in these external dimensions between student nurses and clinic women of the same pelvic type and it is our opinion that to attempt to predict pelvic type on the basis of external dimensions in individual cases is quite hazardous.

7. The high incidence of dolichopellic and mesatipellic pelves among the largest women of both groups suggests the possibility that nutritive and other factors which make for the attainment of maximum normal growth tend to prevent anteroposterior flattening of the pelvis, such as is noted in the brachypellic and platypellic types.

8. Finally, our observations indicate that the type of pelvic inlet can be determined in the intact living women only by roentgenographic means.

THE ESTIMATION OF PELVIC CAPACITY

Modern methods for measuring and estimating pelvic capacity may be divided into two groups: (1) external and internal palpatory methods; (2) roentgenologic methods. An outline-chart of the procedures used in our clinic is shown in Figure 9. This is known as the Pelvic Estimation Chart, and it is filed with each patient's prenatal record in order to be available as soon as she enters the hospital for delivery. It is my conviction that such data, together with a brief comment, will furnish information concerning pelvic capacity which is adequate for all ordinary obstetric purposes. It might be useful to consider in order some of the details of examination that are noted in this outline.

External Measurements. As stated before, the information gained from these external measurements should be considered solely from the standpoint of the guide post. Small external measurements are usually found in patients with pelves of small capacity, but the reverse is by no means true. When the external conjugate diameter is relatively shortened, the probability of anteroposterior shortening of the pelvic inlet should be considered. While the interspinous and intercristal diameters give but little information concerning pelvic capacity, nevertheless their relation to each other may be of significance. In certain cases of rachitic pelvis, owing to the outward flare of the iliac bones, the normal relationship of these two measurements may be reversed, the intercristal diameter being shorter than the interspinous.

New Haven Hospital and Dispensary — Pelvic Estimation Chart

Name_____ Date_____ No _____

External Measurements	Spines cm. Crests cm. Ext. Conj. cm.

External
Measurements   Spines   cm.   Crests   cm.   Ext. Conj.   cm.

     Pubic Arch   Wide   Average   Narrow

     Rami   Arcuate   Straight

     Bituberal   cm.   Post Sag.   cm.

Rectal and Vaginal Examinations   Ischial Spines   Large   Average   Small

     Side Walls   Parallel   Convergent

     Sacrum   A. P. Curve   Concave   Straight   Convex

           Lat. Curve   Concave   Straight   Convex

     Notch   Wide   Average   Narrow

     Coccyx   Movable   Immovable

           Straight   Projects anteriorly

     Diag. Conj.   cm.   Not reached

     Conclusions

                                                    Examiner

Roentgen Measurements

INLET		MIDPLANE		OUTLET	
A. P. ___cm.		A. P. cm.		Bituberal cm	
Trans. cm.		Trans. cm.		P. S. cm.	
P. S. cm.		P. S. cm.			

Pelvic Contours   Inlet   Symmetrical   Asymmetrical

     Ant. Segment   Wide   Average   Narrow

     Notch   Wide   Average   Narrow

Type of Pelvis

Comment

                                                    Examiner

FIG. 9. Pelvic estimation chart.

The Pubic Arch. Palpation of the pubic arch with the thumbs is an important procedure. By this means the course of the pubic rami may be estimated. These bones form a typical arch in the female
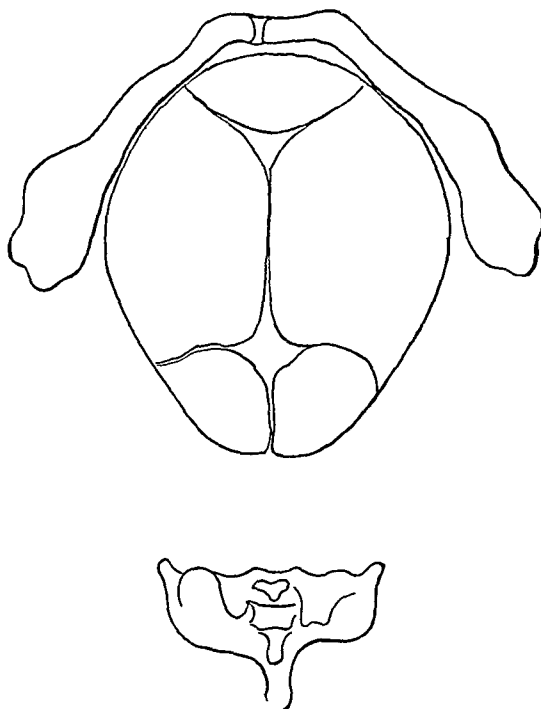
FIG. 10. Showing female type pelvic arch with fetal head fitting under symphysis. (From Thoms' "The Obstetric Pelvis," Williams & Wilkins.)

type, or their course may be straight, forming an inverted V in the male type. In the latter instance, the tubera ischii are usually not so widely separated and a funnel type of pelvic outlet may exist. In this latter type of pelvis it is obvious that in the process of birth the fetal head cannot fit up under the symphysis, and perineal lacerations and delayed labor may be frequently encountered. (Figs. 10 and 11.)

The width of the pubic arch may be roughly determined by palpatory methods. Some observers use the closed fist as palpatory mechanism to ascertain whether the arch is wide, average, or narrow. The bituberal diameter of the outlet may be measured with satisfaction by using the outlet pelvimeter, which was devised by the author some years ago. (Figs. 12 and 13.) The important thing

is to determine just where the end points of such measurement should rest on the tubera ischii. I find that there is no consensus of opinion in this matter. My own practice is to attempt to determine in each
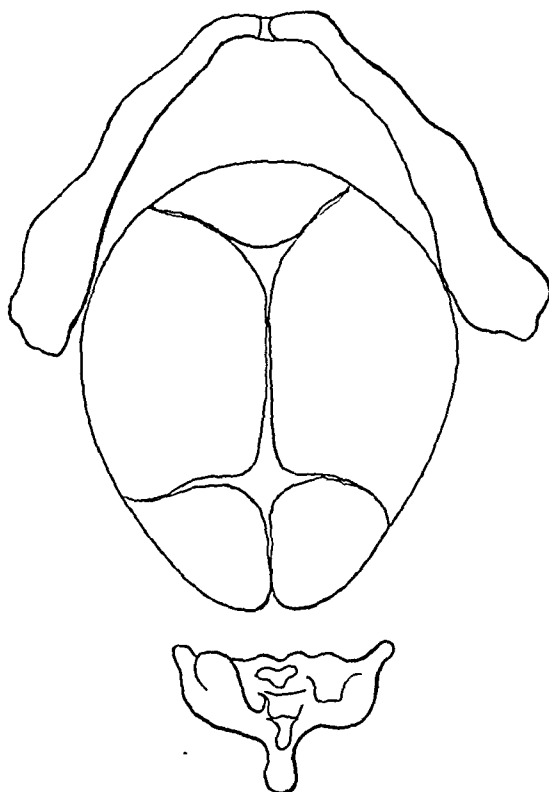
FIG. 11. Showing male type pelvic arch with fetal head forced posteriorly toward sacrum. (From Thoms' "The Obstetric Pelvis," Williams & Wilkins.)

case at which point on the inner edge of the backward curve of the tubera the fetal head might touch if impingement took place. As a rule these points are located at the lowest portion of the inner edge of the tubera ischii, assuming the pelvis to be resting with its tubera in contact with an horizontal surface such as obtains in the upright sitting position. The posterior sagittal diameter of this plane has been previously described and may be measured as shown, with the Thoms pelvimeter. The important thing in this latter procedure is to have the patient's hips well over the edge of the table so that the lower posterior surface of the sacrum may be easily palpated. As this diameter may be also measured roentgenologically, the two readings should check within reasonable limits.

Ischial Spines and Side Walls. These eminences are easily palpable on both rectal and vaginal examination. The interspinous diameter also may be roughly estimated, but the chief object of
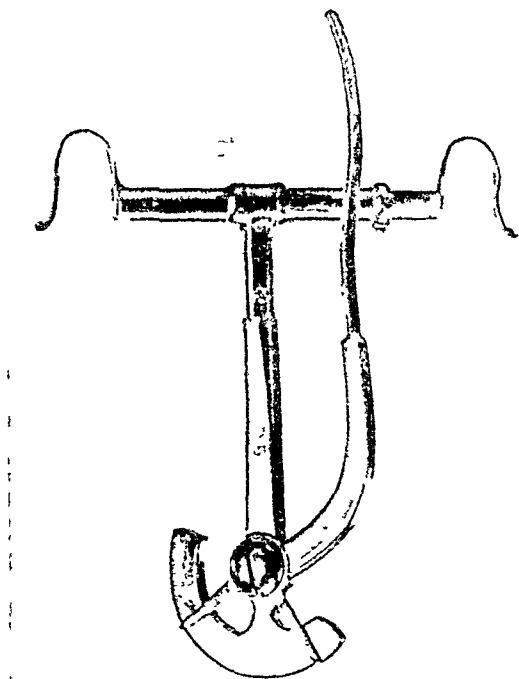
FIG. 12. The author's outlet pelvimeter. Half size.
(From Thoms' "The Obstetric Pelvis," Williams
& Wilkins.)

the internal examination is to determine the size and prominence of the spines in terms of large, average, and small. The male type of ischial spine conformation is almost universally large and prominent. The lower side walls of the pelvis are readily palpated, especially where there is narrowing or convergence such as is seen in male pelves and in certain pelves of the dolichopellic type.

The Sacrum. Careful rectal and vaginal palpation will determine a good deal of information concerning the anteroposterior and lateral curves of the sacrum. The former may be investigated by sweeping the examining finger upwards and downwards over the anterior surface of the sacrum and the lateral curve determined by a side to side motion over this surface. In this way we may find

out whether these surfaces are concave, straight, or convex, which may be important information in the diagnosis of abnormalities of the sacrum due to rachitic influence.
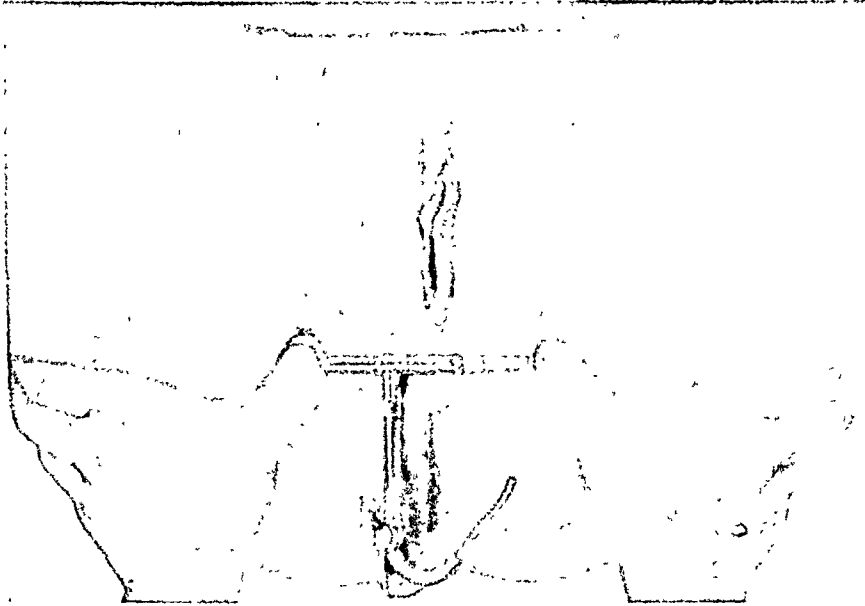
FIG. 13. Measuring of transverse diameter of outlet. (From Thoms' "The Obstetric Pelvis," Williams & Wilkins.)

A   B
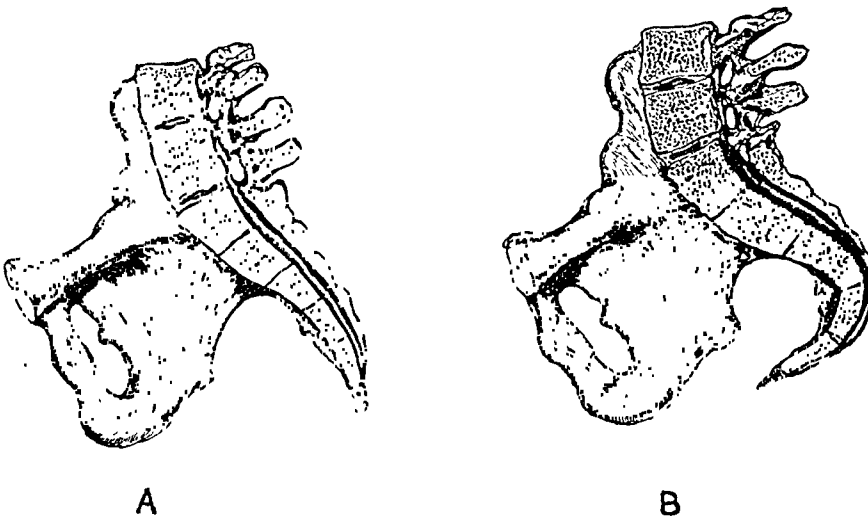
FIG. 14. Two types of abnormal sacral curvature. A, sacrum straightened out. May be entirely convex from above downward. B, abnormal concavity of sacrum. (From Thoms' "The Obstetric Pelvis," Williams & Wilkins.)

The Sacrosciatic Notch. This excavation may be palpated both vaginally or rectally. The latter procedure is probably the more useful. The strong ischiosacral ligaments may be readily felt and a

good idea of the character of the lower part of the notch be elicited, especially when abnormal narrowing is present.

   The Coccyx. This bone is readily felt on vaginal and rectal
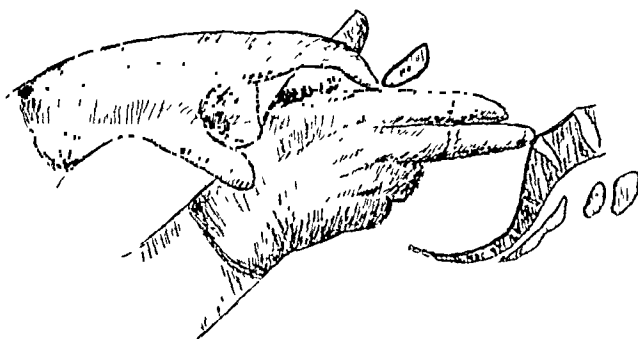
FIG. 15. Measuring the diagonal conjugate diameter. (From
Thoms' "The Obstetric Pelvis," Williams & Wilkins.)

examination, and its relative movability established. If its position is a continuation of the downward sweeping curve of the anterior sacral surface it is said to be straight. If, however, the coccyx projects anteriorly, forming an angle with the tip of the sacrum, this is noted. The possibility of delayed labor due to forward malposition of the coccyx should not be overlooked.

   The Diagonal Conjugate Diameter.   The directions for obtaining this diameter are essentially as follows (Fig. 15): With the patient in the lithotomy position and prepared for vaginal examination, the examiner raises one knee by placing the foot upon a small stool. Then, sinking the elbow and resting it against the knee, the vaginal examination is made, using two fingers. The middle fingertip is pressed in the direction of the sacral promontory by means of steady pressure against the perineum. As soon as the middle fingertip touches the promontory, the internal hand is raised against the lower border of the symphysis. At this point the index finger of the outside hand rests on this latter point and the internal hand is withdrawn. The distance from the middle fingertip to the point marked by the index finger of the other hand is measured by calipers or tape to obtain the length of the diagonal conjugate diameter. DeLee states "After guessing at the inclination of the pubis and allowing for height, one deducts 1½ or 2 cm. or more, and thus approximates the length of the C.V. (true conjugate). Numerous fallacies underlie this reading, the compression of the fingertip, the bending of the joints, the slipping of the finger from the promontory, errors in

estimating the height, thickness, and angle of the pubis, etc."
(Fig. 16.) To these I would add that in certain individuals, particularly primigravidae, the examination may be difficult because of
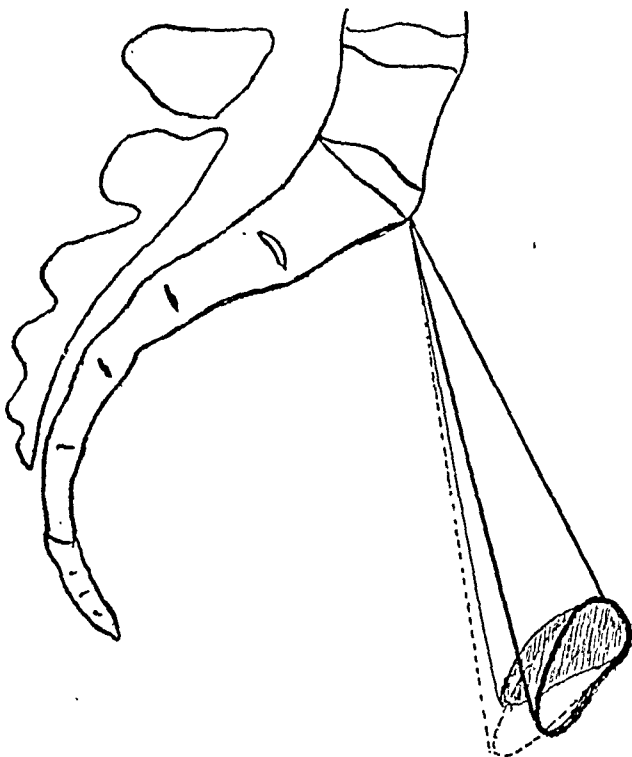
FIG. 16. Showing the effect of forward displacement and
elongation of symphysis on the length of the diagonal
conjugate diameter. (From Thoms' "The Obstetric
Pelvis," Williams & Wilkins.)

the pain experienced by the patient due to the rigidity of the perineal
soft parts. More important than any of the above fallacies, is the fact
that in many pelves the sacral promontory is so high above the
posterior point of convergence of the iliopectineal lines that the
reading becomes practically valueless for obstetrical calculation.
In these cases, as has been pointed out previously, the plane of the
inlet or true obstetrical inlet is not encroached upon, or influenced
by, the promontory of the sacrum; hence any anteroposterior diameter using the promontory as an endpoint may be misleading.

After these objections, one may wonder why any attempt at all is
made to measure this diameter. The author's feeling is that while
making the routine vaginal examination to elicit other information,
reasonable attempts should be made to reach the promontory also.
If it is accessible, this measurement may be determined, but if it is

not reached readily, pronounced attempts to do so should not be carried out and the designation "not reached" should be entered on the outline chart. So many factors, however, result in an inability
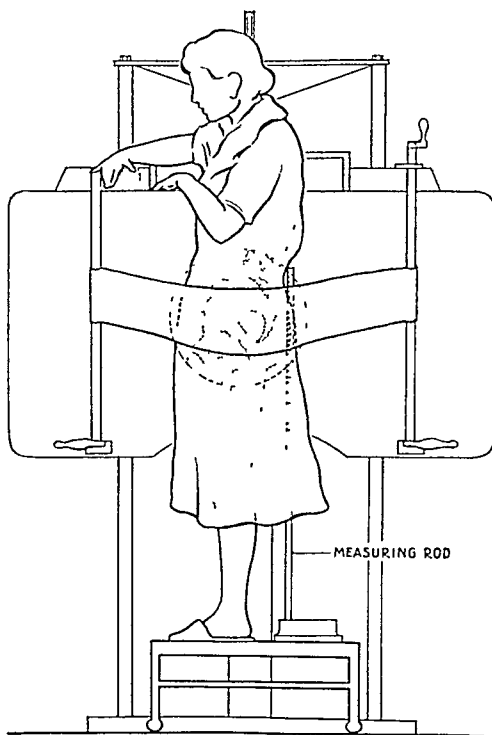
FIG. 17A. Position of the patient for lateral pelvimetry. (From Thoms and Wilson, in *Yale J. Biol. & Med.*, 10: 437, 1938.)

to reach the sacral promontory that by no means should the designation "not reached" be construed as indicating that the antero-posterior diameter of the pelvic inlet is adequate for obstetrical purposes.

Conclusions. Under this heading the examiner briefly notes any abnormalities or variations from the normal, also his opinion of the adequacy of the pelvis in its entirety.

ROENTGEN METHODS FOR PELVIMETRY

The author's first communication on the subject of pelvimetry appeared in 1915, and in 1922 the results of his first experiments with Roentgen pelvimetry appeared. From then until the present, the results of his experience in applying Roentgen methods to clinical problems have appeared from time to time. In 1938, in collaboration

with H. M. Wilson, certain Roentgen methods were described which are useful for routine obstetrical pelvimetry, and these techniques with slight modifications may be described again here.
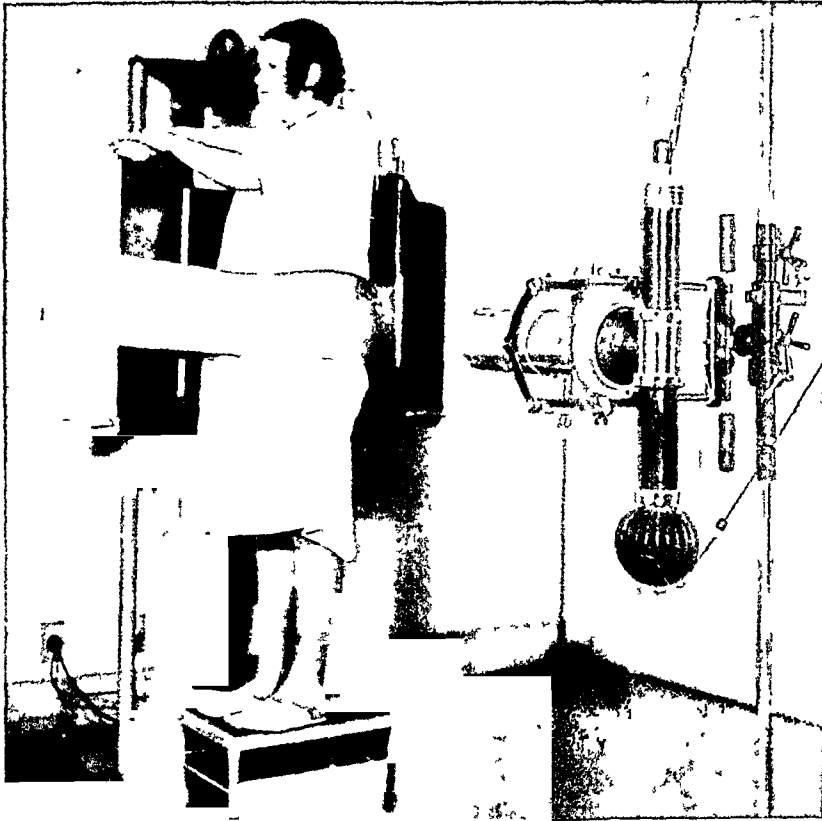
FIG. 17B. Position of the patient for lateral pelvimetry. (From Thoms and Wilson, in *Yale J. Biol. & Med.*, 10: 437, 1938.)

For routine purposes we take two views: one of the pelvic inlet by the so-called grid method, and one lateral projection. It has become obvious to us that an adequate survey of the pelvis for obstetrical purposes is not possible without the employment of both techniques. Furthermore, inasmuch as the mensuration of the antero-posterior diameter of the pelvic inlet may be determined in both views, each procedure becomes an excellent check on the accuracy of the other. Our routine is to take the lateral projection first, and this technique may be described as follows:

1. The patient removes her clothing, putting on heelless slippers and the usual hospital bed-gown open at the back.

2. She is placed standing in front of an erect Bucky diaphragm or an adjustable cassette changer, such as is used for chest work, with

either the right or left lateral aspect of the body toward the target. (Fig. 17.) The arms are folded across the chest.

   3. The target film distance is 40 inches and the target is centered
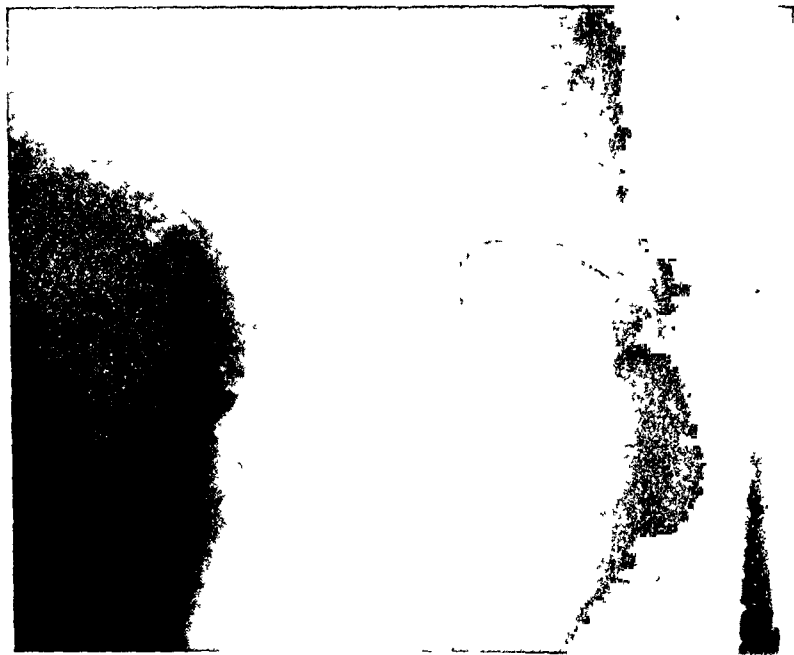
FIG. 18. Lateral roentgenogram at term. Fetal head shown above the pelvic inlet. Evidence of slight disproportion is present. Corrected centimeter scale at side of film, by means of which all anteroposterior diameters may be measured. (From Thoms and Wilson, in *Yale J. Biol. & Med.,* 10: 437, 1938.)

at a point on the external conjugate diameter one-third the distance from the symphysis pubis to the depression under the fifth lumbar vertebra. A 60 inch target film distance may be used, employing a Lysholm grid instead of a Bucky diaphragm, as shown in Figure 17. The shorter distance with the Bucky diaphragm seems to give somewhat better definitions in our hands.

   4. A binder is placed around the patient and attached to the cassette changer to insure further steadiness during the exposure.

   5. Before the exposure is made, an upright metal rod (lead and iron) with a centimeter scale perforated in a lead strip is placed posterior to the patient close to the fold of the nates. This rod is somewhat similar to that described by Weitzner for use in lateral horizontal position.

6. The time of exposure varies with the thickness of the patient, all other factors being constant; in general the time is from twelve to fifteen seconds.

Comment on Lateral Technique. When developed and viewed, the following landmarks should be readily identified: anterior and posterior borders of the symphysis pubis, acetabula, ischial spines, ischial tuberosities, the lower lumbar vertebrae, the promontory and anterior surface of the sacrum, and the sacrosciatic notch. (Fig. 18.) On one edge of the film may be seen the shadows cast by the perforations giving corrected centimeters in the sagittal plane of the patient. By means of calipers, using this scale, any diameters in this plane may be measured.

CHART OF TECHNIQUE FOR LATERAL PELVIMETRY

Kvp.. 80
Ma... 30
Dist... 40 inches
Bucky diaphragm... Yes
Time.. Chart
Screens... HiSpeed
Medium sized cone

THICKNESS TIME-CHART

Centimeters	Seconds	Centimeters	Seconds
27	9½	33	15
28	10	34	17
29	11	35	19
30	11½	36	21
31	12	37	23
32	13	38	25

The advantages of this lateral technique may be summarized as follows:

1. All the anteroposterior diameters of the bony pelvis may be measured, including those of the pelvic inlet, midpelvis, and inferior strait. The anterior and pelvic inlet diameters of the important midpelvic plane are readily determined.

2. The contours of the anterior surface of the sacrum may be studied, a matter of importance in the recognition of sacral abnormalities, especially those due to the influence of rickets.

3. When lateral roentgenograms are made at term or in labor the relation of the presenting part to the superior strait may be studied with advantage. Since early in labor, in the majority of

instances, the fetal head approximates the L.O.T. or R.O.T. position, it will be seen that it is possible to measure also the all important biparietal diameter of the fetal head. In cases where the so-called trial of labor is being given and the patient is having frequent uterine contractions it may be more convenient to use the lateral horizontal position. When this is done we use the same distance, reduplicating as far as possible the relationships which we have used for the same patient in the erect lateral projection.

4. In certain instances where diagnosis is doubtful respecting the presence of multiple pregnancy or fetal abnormality, lateral roentgenograms may give more information than that obtained by the usual anteroposterior technique.

The advantages of this present modification appear to be as follows:

1. Decreased distortion. The use of a 40 or 60 inch target film distance for projection of the sagittal plane lessens the distortion due to divergence of the x-ray beam. This is especially true in large subjects whose lateral pelvic diameter results in a long object film distance. The erect posture also decreases distortion resulting from rotation of the pelvis on its vertical axis and tilt on its anteroposterior axis. With the subject horizontal on a flat table it is difficult to secure good superimposition of the shadows of the two innominate bones. The erect position not only corrects this, but permits a more satisfactory projection of the lumbosacral relationship. The sag of the lumbar spine, due to the disproportion between the pelvic and shoulder diameters when the patient lies on her side, is not met in the erect position.

2. Corrected centimeter scale. The projection of a centimeter scale corrected for the sagittal plane of the individual pelvis appears on the edge of the finished film. The obstetrical conjugate is determined by selecting a point on the cortical white line of the posterior wall of the symphysis 1 cm. below the superior border and measuring with calipers the distance between this point and the upper sacrum at the point where the iliopectineal lines meet.

3. Simple equipment. The erect posture in lateral pelvimetry makes possible a long target film distance. No special apparatus is required other than the perforated opaque scale and the Bucky diaphragm or Lysoholm grid.

The centimeter grid method for pelvic inlet pelvimetry may be described in its essentials as follows:

1. The patient is placed on the roentgenographic table in a semi-recumbent position which is maintained by a backrest shown in Figure 19. (The lower third of this rest is cut away in order that
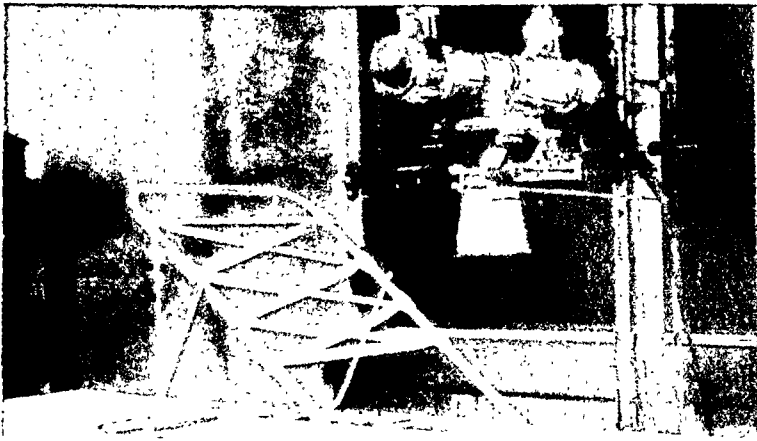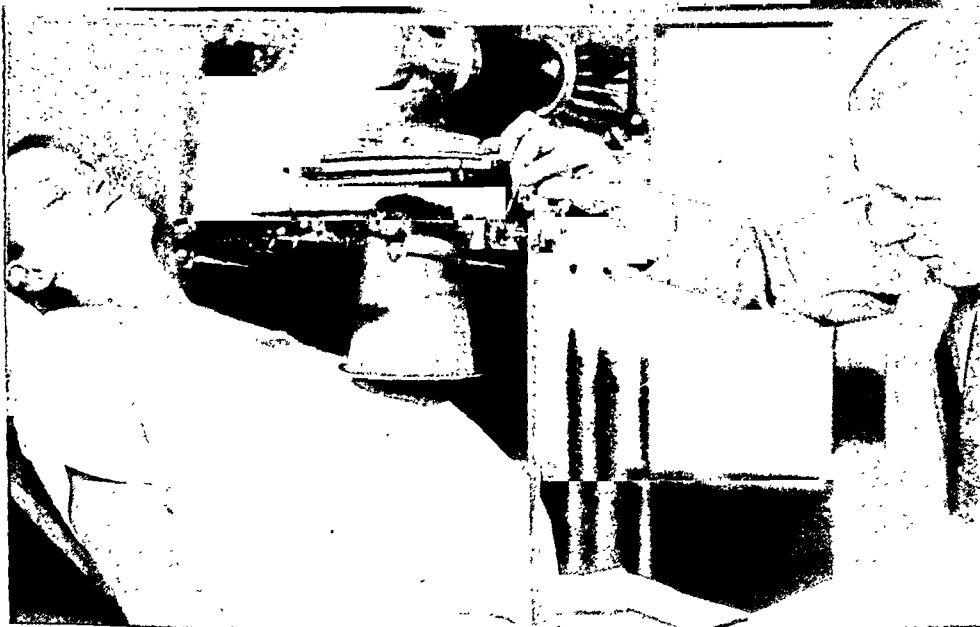
FIG. 19. Back rest used for pelvic inlet pelvimetry.
FIG. 20. The superior border of the symphysis is located. (From Thoms and Wilson, in *Yale J. Biol. & Med.*, 10: 437, 1938.)

the lower part of the patient's back may be exposed.) In placing the patient in position we endeavor to make the pelvic inlet of the

pelvis as nearly horizontal as possible, with the patient in a comfortable, unstrained position.

<div align="center">CHART OF TECHNIQUE FOR SUPERIOR STRAIT PELVIMETRY</div>

Kvp	80
Ma.	30
Dist	30
Bucky Diaph	Yes
Time	Chart
Screens	HiSpeed
Medium cone	

<div align="center">THICKNESS TIME-CHART</div>

Centimeters	Seconds	Centimeters	Seconds
24	1	32	8
25	2	33	9
26	3	34	12
27	3½	35	14
28	4	36	17
29	5	37	20
30	6	38	23
31	7		

2. The level of the pelvic inlet above the sensitive film is established as follows: (a) By means of calipers the vertical distance is measured from some point on the table top to a point on the anterior surface of the symphysis pubis 1 cm. below its superior border, or this point in space may also be determined by measuring downward with a tape from some point on the tube-stand. (Fig. 20.) (b) By means of calipers the distance is determined from the interspinous space between the fourth and fifth lumbar vertebrae, as determined by palpation, and the table top. (Fig. 21.) For practical purposes, an imaginary line drawn between the posterior point on the body and the point on the upper and anterior surface of the symphysis will bisect the plane of the pelvic inlet.

3. The tube is centered in the midline about 6 cm. posterior to the upper border of the symphysis and the exposure is made.

4. The patient is removed from the table, the tube and exposed film remaining *in situ*.

5. The centimeter grid, a lead plate with perforations exactly 1 cm. apart, is introduced into the same plane as that previously occupied by the pelvic inlet (Fig. 22), as determined by the caliper readings and the measuring tape, and a second (flash) exposure made on the previously exposed film.

FIG. 21. The interspace between the fourth and fifth lumbar vertebrae is located.
FIG. 22. The centimeter grid is placed in the plane formerly occupied by plane of the pelvic inlet. (From Thoms and Wilson, in *Yale J. Biol. & Med.*, 10: 437, 1938.)

On viewing the developed film an outline of the pelvic inlet is shown, together with shadows produced by the perforations in the lead plate. (Fig. 23.) The distance between these projected dots

FIG. 23. Outline of pelvic inlet. Distance between dots represents centimeters in this plane. The anteroposterior diameter should check with that of the lateral projection. The bispinous diameter also may be measured in this film by correcting for the plane in which these processes rest (see text). (From Thoms and Wilson, in *Yale J. Biol. & Med.*, 10: 437, 1938.)

represents centimeters in the plane of the pelvic inlet. Not only may the anteroposterior and transverse diameters of this plane be read directly, but a reduced outline of the pelvic inlet may be drawn on centimeter paper and this record filed with the prenatal record. The latter procedure is but a matter of reduction and the simple apparatus which we use for this purpose has been recently described.

    Comment on Centimeter Grid Technique. For obstetrical and investigative purposes we have no reason to doubt the accuracy of our results. On numerous occasions we have performed experiments,

both with dried pelves and in the living woman, in which our results have been checked at laparotomy. Interesting in this latter connection is the recent communication of Dr. E. A. Schumann, who, in
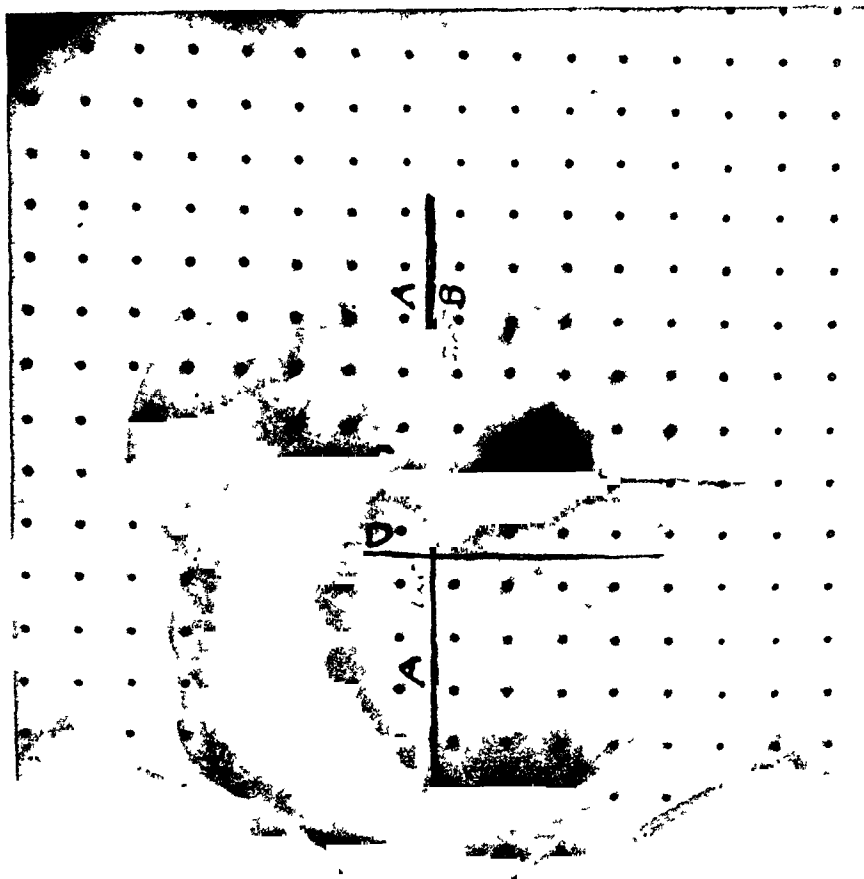
FIG. 24. Film of pelvic inlet. Lines indicate cardinal diameters: A, anteroposterior diameter of inlet. B, posterior sagittal diameter of inlet. C, transverse diameter of inlet. D, transverse diameter of midplane.

1936, measured fifty women directly at laparotomy and observed: "In order to determine the accuracy of roentgenologic measurement, a group of the foregoing patients were subjected to measurement by the technique of Thoms, and it was extremely gratifying to learn that the diameters of the inlet as determined by direct intra-abdominal measurement corresponded in every instance with those obtained by this technique, the variation being constantly less than 2 mm."

The question naturally arises as to whether the posterior point on the patient's body (i.e., the interspinous space between the fourth

and fifth lumbar vertebrae) is too indeterminate for the placing of the grid in the plane of the pelvic inlet. Our experiments with dried pelves have shown us that this posterior point may vary as
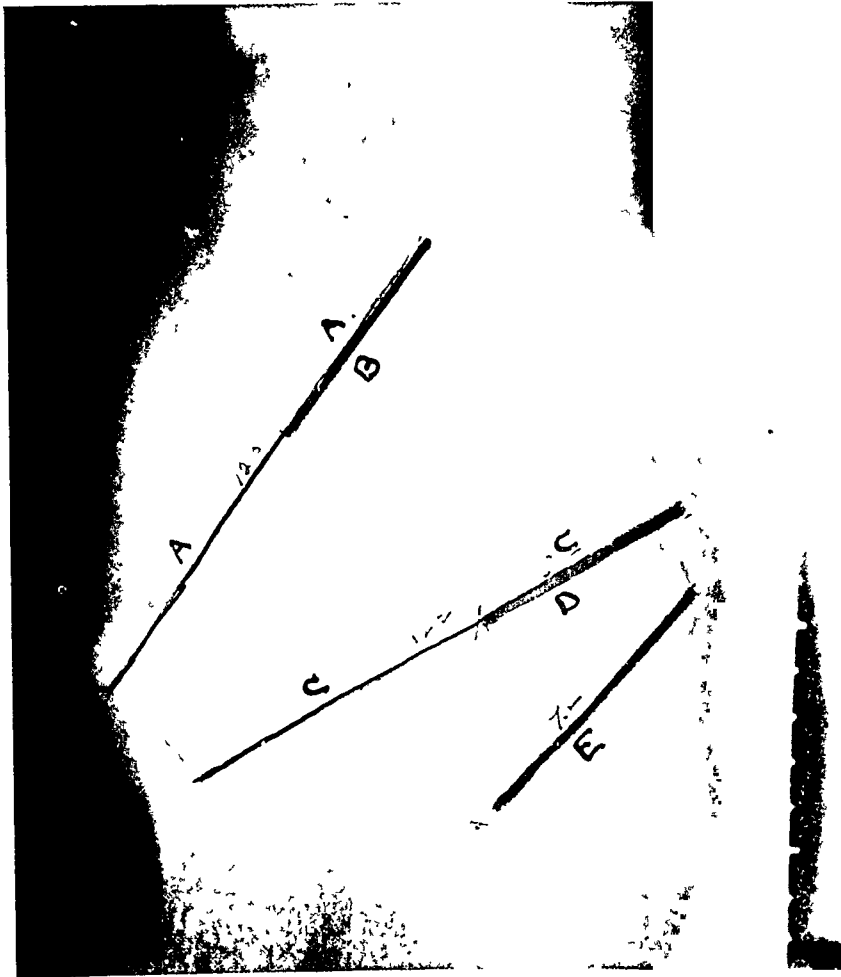
FIG. 25. Film of lateral pelvis. Lines indicate cardinal diameters. A, anteroposterior diameter of inlet. B, posterior sagittal diameter of inlet. C, anteroposterior diameter of midplane. D, posterior sagittal diameter of midplane. E, posterior sagittal diameter of outlet.

much as 2.5 cm. above or below the ideal situation, without measurably affecting the mensuration as recorded on the film. In other words, there is a leeway of at least 5 cm. in determining this point. In any event, the lateral projection should leave no doubt as to the true anteroposterior diameter of the pelvic inlet and the centimeter grid projection should check this within 2 mm.

Furthermore, it is entirely possible, by developing and viewing the lateral projection before placing the grid in position for exposure, to determine exactly its ideal position. This occasionally may be necessary for exact results in rare instances of marked sacral deformity where a double promontory or other malformation places the obstetrical conjugate in a plane much different from that of the anteroposterior diameter of the superior strait. When it is desired to regulate the placing of the grid in this manner, this may be accomplished by projecting a line on the lateral film from the posterior border of the symphysis through the plane to be measured. With the calipers (using the corrected scale) we measure downward at a right angle to this line to the tip of the spinous process of the last lumbar vertebra as seen in the projection. With the patient in the sitting position we measure the distance of this spinous process above the table top and the data are complete for the exact placing of the grid. However, for routine purposes we are confident that this refinement in technique is not necessary, for when the lateral projection is used as a check, any discrepancy of the anteroposterior diameter in the centimeter grid projection will be manifest.

Summary. Among other features which impress us in using these methods may be mentioned the fact that the roentgenograms so obtained are readily interpreted by both the obstetrician and the roentgenologist. The simplicity of these methods, using but two 10 × 12 films and consuming a minimum of time for the technique, reduces the expense of Roentgen pelvimetry to a very reasonable sum. A further reduction in overhead expense is possible by grouping patients to be done at one time. The use of the lateral technique before and during the so-called "trial of labor" is of great advantage in studying the cephalopelvic relationship.

It is important to mention again the mensuration of the diameters of the midpelvis and pelvic outlet, since these portions of the pelvis play a very definite part in dystocia. The distance between the ischial spines may be measured in these films by first noting the distance of these processes below the anteroposterior diameter on the lateral film and then measuring the interspinous distance on the inlet film, correcting by means of a table showing the spread of the rays at the level of the plane in which these processes rest.

CONCLUSION

A little over two years ago the author published a paper dealing with the uses and limitations of Roentgen pelvimetry, and much

that was said at that time bears repetition here. The experience which has come from the use of newer methods of measuring the pelvis has taught us that we are in a transitory period from the general use of older and somewhat approximate methods to the adoption of newer and more accurate procedures. These newer methods have not only furnished new concepts as to the female bony pelvis but also more knowledge of cephalopelvic adaptation during the process of birth.

In the present monograph, which concerns itself chiefly with the estimation of pelvic capacity, it would be helpful indeed to consider in further detail certain clinical relationships which pelvic capacity may affect. At the present time, however, it may be said that many such important relationships are not completely established and may become so only through further clinical experience. The reason for such discrepancies is apparent when we consider the many factors that are concerned in the process of birth. I have previously commented upon this matter as follows. "The birth canal is composed not only of the bony structures but of soft parts, and the fetus also is composed in the same way. Mensuration of these bony structures therefore is not an entirely complete survey of the birth passage and its passenger. The forces which are concerned in labor are subject to influences which render them both voluntary and involuntary, and it would appear that much of this influence is hormonal in character. In addition, the influence of such factors as race, constitution, disease, emotion, and pain all share in the complex process. The usefulness of the knowledge of the dimensions of bony structures of the pelvis and fetus, therefore, has certain limitations, and to draw definite conclusions as to the outcome of labor without considering the whole picture is not only hazardous but unscientific. In other words, the greatly useful knowledge which these Roentgen methods has given us must be properly assimilated with our greatest therapeutic weapon, namely, clinical experience. It is not enough to visualize the pelvis and fetus in space by means of roentgenograms. Indeed it is easy for the inexperienced and enthusiastic to be sidetracked into false security if too much emphasis is given to Roentgen information so obtained. Roentgen methods are of great value, but they must be properly interpreted."

Again it is the author's wish to emphasize the importance of the routine use of the measures recommended in primigravid women. An unsuspected midplane or an outlet contraction may be present in the primigravid patient at term with adequate inlet capacity in whom

the fetal head is engaged, and there is no way of accurately determining such occurrence without Roentgen means. Furthermore, the occurrence of wide variations of pelvic form in presumably normally formed individuals is definitely a matter of concern to the obstetrician, and only by Roentgen investigation may these variations be established. With regard to the use of these methods in the presence of pelvic contraction, comment seems hardly necessary. Dippel in a recent comment upon this question has ably stated the correct scientific viewpoint. "It may be argued," he writes, "that there are so many factors in labor, namely, the size of the baby and the character of the uterine contractions, that an approximation is enough. We do not agree with this viewpoint. The very fact that these other factors are difficult to evaluate accurately makes it all the more important, in cases of contracted pelvis, that we should have as precise information as possible concerning the one factor that we can measure with precision."

In conclusion, the author wishes to emphasize that at the present time we possess methods for measuring the female pelvis which are quite adequate for obstetrical purposes and by their routine use we are bound to enrich our knowledge and experience for the betterment of scientific obstetrics.

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